Sixteenth Annual Meeting — American College of Chest Physicians
St. Francis Hotel, San Francisco, California, June 22-25, 1950

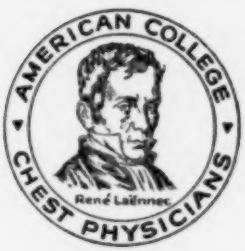
VOLUME XVII NUMBER 2

DISEASES

of the

CHEST

OFFICIAL PUBLICATION



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DISEASES *of the* CHEST

OFFICIAL PUBLICATION

OF THE

AMERICAN COLLEGE OF CHEST PHYSICIANS

EXECUTIVE OFFICE
500 North Dearborn Street, Chicago 10, Illinois
MURRAY KORNFELD, *Managing Editor*

VOL. XVII FEBRUARY 1950 NUMBER 2

CONTENTS:

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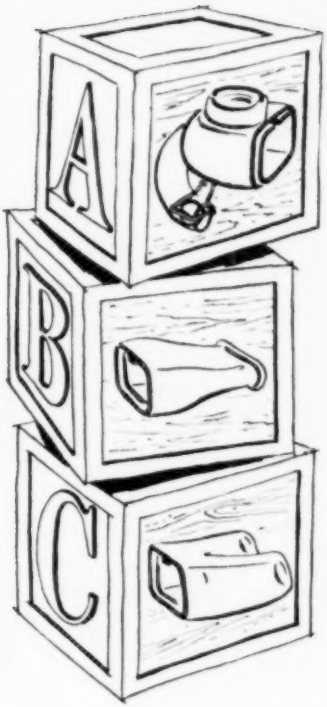

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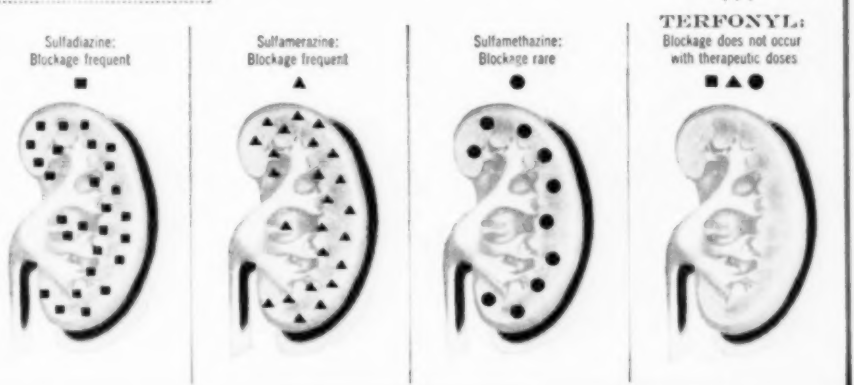

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DISEASES *of the* CHEST

VOL. XVII FEBRUARY 1950 No. 2

Bronchial Obstruction*

CHEVALIER JACKSON, M.D., F.C.C.P.
Philadelphia, Pennsylvania

The magnitude of the subject, including as it does, hundreds of problems in physiology and biochemistry as well as in nearly all medical and surgical pulmonary diseases, precludes consideration of more than a few phases within the limits of one paper. The revelations of the bronchoscope during the first half of this century have contributed so much toward solution of some of the just mentioned problems, that a review and formulation of the bronchoscopic phases of the subject seems desirable. This paper is an effort toward that objective.

Incidence: Bronchial obstruction, temporary or prolonged, is a universal pathologic occurrence, common among the newborn and many times in later life. The terminal phase of most slow deaths with the patient on his back is asphyxia from drownage of the patient in his own secretions. Incipient bronchial obstruction attends every acute infection of the lower air passages. In most of the cases, physiologic peroral drainage, by ciliary wafting, tussive squeeze and bechic blast, expels the partially obstructive material before it accumulates and undergoes changes that make it occlusive or nearly so.[1] When the quantity and cohesiveness of the exudates overhelm the normal tussive expulsive mechanism, when the vital cough reflex on which the activation of this drainage system depends, is suppressed by antibechics drugs, toxic conditions, exhaustion, prolonged surgical operation or similar producers of stagnation, bronchial obstruction becomes a serious and often fatal pulmonary complication. No record of bronchial obstruction appears in vital statistics. There are however, no end of clinical observations, including bronchoscopic, to prove that it

*Presented at the Fifteenth Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 3, 1949.

is a chief and determinant factor in morbidity and mortality in the pulmonary diseases recorded. The patients who survive an acute bronchial obstruction may die later of sequential bronchial or pulmonary disease yet no record of the bronchial obstruction ever appears in vital statistics.

Prior to the advent of the bronchoscope the only objective knowledge of bronchial obstruction was afforded by the cadaveric terminal conditions present in the motionless bronchi as revealed at necropsy. The bronchoscope has revealed to the eye pathologic conditions present in the living moving bronchi; conditions that exist long prior to the terminal stage revealed at necropsy. The endoscopic image, and the constant shifting of the picture presented, strikes the observer experienced in postmortem appearances as a newly discovered pathologic mechanism.

A brief résumé of established facts regarding bronchial obstruction will conduce to clearness and brevity.

Valvular Obstruction

It is a platitude to say that cadaveric pathology is the foundation of medical science. In relation to bronchial obstruction, however, necropsy does not show the important factor of bronchial movement. Movement relates the bronchi to that part of science known as mechanics, which treats of the action of forces on bodies, solid, liquid and gaseous. So long as the bronchi contain only gaseous bodies they are concerned with the laws of pneumatics. When they contain liquid bodies they are concerned with the laws of hydrostatics and hydrodynamics. The presence of solids brings into consideration the fundamentals of mechanics in its restricted sense. Additionally bronchial obstruction is especially concerned with intermediate semisolid bodies which do not flow, particularly secretions and exudates of a high degree of cohesiveness and adhesiveness.[1,2] In the field of applied mechanics, for the control of movements of gaseous and liquid bodies in tubes, mechanical engineers use valvular mechanisms known as (a) stop valves, which arrest all flow in either direction; (b) by-pass valves, which permit a diminished flow in either direction, and (c) check valves, which permit a flow in one direction only; the direction of the flow, however, can be reversed by resetting the valve in a reversed position. Check valves, or one-way valves as they are called by European mechanical engineers, are of two types: (1) The ball check valve, in which a ball, whose escape is prevented by a cage, is raised from its seat by the flow of the tubal contents, but is promptly forced back into its valve seat by any tendency to a reversal in the direction of flow. (2) The type much more commonly used by engineers is the flapper check valve in which a

hinged flapper is lifted from its valve seat by a flow in the desired direction, but is promptly forced back by any tendency to flow in the opposite direction. Both these types of check valves are frequently seen bronchoscopically as represented by a ball-like mass of secretion rising on expiration, permitting exit of air from a lobe or segment, and falling back into a bronchial orifice (or into a funnel-like diminution of lumen in the form of an annular swelling of the mucosa), thus preventing ingress of air to the tributary area, which thus becomes atelectatic. In some cases the ball-like mass of secretion is below an annular swelling and is blown up against the ring-like narrowing at each expiration. This produces emphysema in the tributary area. The flapper mechanism is often seen bronchoscopically as a thick, adhesive, tenacious blob of secretion hinged at one end by adherence to the bronchial wall, the free end rising with expiratory egress of air, flapping down and checking ingress of air to the tributary area on inspiration.

There is a third type of check valve not represented in mechanical engineering. We have always called it the *expansile* check valve.[1,7,8,12,13] Its mechanism is the enlargement of lumen on inspiration and the diminution of lumen at the start of expiration. Bronchoscopic study revealed that in case of an endobronchial exogenous foreign body, the enlargement of lumen on inspiration permitted air to pass downward through a chink opened between the tumor or the foreign body and the wall of the inspirationally enlarged bronchus. It was also observed that, *immediately at the start* of the expiratory phase, this more or less crescentic chink, visible at the maximum luminal enlargement, promptly disappeared, and that the wafting downward of marginal secretions noted on inspiration was not reciprocated by an upward bubbling of secretions. This objective evidence of an air trap mechanism was observed in cases in which the physical signs showed a hyperresonance over the corresponding lung, and the roentgen ray showed an emphysematous radioparency of the same region. Subsequent observations showed that this mechanism in some cases involved only one lobe, in other cases only a segment of a lobe. Comparative clinical studies by Willis F. Manges, Thomas McCrae and Chevalier Jackson of the abundant material of the bronchoscopic clinic, developed the knowledge of the phenomena of obstructive emphysema and obstructive atelectasis to a great degree of practical usefulness, not only as to the presence of a radioparent exogenous foreign body or tumor, but also as to its location in the tracheobronchial tree.[7] It was discovered bronchoscopically that though all these check-valve mechanisms, (a) the expansile, (b) the flapper and (c) the ball-valve, were operative in cases of

exogenous foreign body in the tracheobronchial tree, they were of enormously greater frequency in cases of endobronchial masses of endogenous substances, such as crusts, sequestra and viscid and membranous exudates.[7,13,15] It was often observed that these check-valve mechanisms, by converting the normal reciprocating inflow and outflow of air produced by the respiratory action of the thoracic cage into a one-way flow, resulted in emphysema when the set of the valve pumped air into a lung or lobe, atelectasis when the set of the valve pumped air out of a lung or lobe. These discoveries added to our knowledge of bronchial obstruction.[1,3-6] Prior to that time the only known bronchial obstruction was static partial or total occlusion, discovered at necropsy. When a bronchus is completely occluded so no air can enter, the residual air in the tributary pulmonary area is presumably absorbed by the circulating blood and the area becomes atelectatic. Though this process of production was often observed it was also frequently noted that the area tributary to a valvular obstruction could be pumped empty quickly without waiting for the slower process of absorption by the circulation.[7] (For clearness it may be stated that it is fundamental to commercial pumps that the mechanical difference between a force pump and an exhaust pump is simply the set of the check valve. Any reciprocating force pump can be changed into an exhaust pump by reversing the check valve). The mechanism of valvular bronchial obstruction have been illustrated in drawings, and the bronchoscopic appearances have been shown in color, in the publications of the bronchoscopic clinic.[2,7,10,12,15]

Etiology of Bronchial Obstruction: The cause of bronchial obstruction may be anything that interferes obstructively with ventilation and upward drainage of the lung. Obviously this includes not only local conditions in the lower air passages but also disorders of the central and peripheral innervation on which the cough reflex depends.

Physiologic Basis: The cough reflex is the "watchdog of the lungs,"[2] constantly on duty to drive out any exogenous or endogenous intruder from the lumen of the lower air passages. Normal peroral pulmonary drainage is by (1) ciliary wafting; (2) tussive squeeze and (3) bechic blast.[1] Fundamentally, anything that lessens these physiologic mechanisms promotes bronchial obstruction. From the purely mechanical viewpoint the first factor is diminution of normal lumen. Any degree of diminution of lumen, however slight, is an element of obstruction. Another mechanical factor is the clogging of the cilia by mucosal swelling and an overload of adhesive inflammatory or other exudates. It is not always realized that the bechic blast cannot remove pathologic secretions from the alveoli or bronchioles, because air below the

obstruction is necessary for efficient expulsion by bechic blast, and there is little or no air below the obstruction.[1,3-6] Though ciliary wafting may help to bring up secretions and exudates to a point at which air for a blast can get below them, swelling as well as the exudates themselves clogs the cilia. Whether ciliary wafting is thus impaired or not, the most powerful agent in supplying the bechic blast with secretions and exudates for expulsion is the tussive squeeze.[1] Another fundamental physiologic factor is constituted by the movements of the tracheobronchial tree. These movements are the rythmic luminal enlargement and elongation alternating 18 or more times per minute with luminal diminution and shortening.[1,2,7,8]

The following tabulation of causative factors relative to bronchial obstruction is from clinical observations in the bronchoscopic clinic:

Tabular List of Causes of Bronchial Obstruction

1. Newborn (non-inflammatory). Intrauterine inspiration of amniotic fluid.
2. Secretions and exudates (essentially endogenous foreign bodies).
3. Infection and inflammation, specific, non-specific, mycotic, mucosal swelling, fibrosis, cicatricial stenosis.
4. Opiates and atropine.
5. Operations: (a) premedication; (b) postoperative medication; (c) prolonged operation; (d) failure to use preoperative and postoperative tracheobronchial aspiration; (e) interference, diaphragmatic; (f) postoperative posture.
6. Endogenous foreign body, sequestra, sloughs, broncholiths. (See also 2).
7. Tumors: (a) endobronchial; (b) peribronchial compressive; (c) malignant; (d) benign; (e) borderline.
8. Adenopathy: (a intrusive; (b) peribronchial; (c) compressive.
9. Allergic conditions, asthma, hay fever, secretions.
10. Non-inflammatory conditions, angioneurotic, paralytic.
11. Exogenous foreign body.
12 Predisposing factors: (a) age; (b) sex; (c) anatomy.

Some of the items tabulated in the foregoing list call for detailed consideration.

Clinical Data on Etiology: The following causes of bronchial obstruction are shown to be common in the records of 45 years of clinical work at the bronchoscopic clinic:

Age: Newborn babies are affected with noninflammatory bronchial obstruction, by inspiration of amniotic fluid at the occasional intrauterine inspiratory movements.[9,10] When birth deprives them

of placenta borne oxygen they asphyxiate by *drownage in meconium*, if they are unable to expel the fluid. Direct laryngoscopic aspiration of the tracheobronchial tree with the smallest silk-woven aspirating tube, followed by *gentle* insufflation of oxygen is a simple life-saving procedure in such cases. (Great care should be taken to avoid distention of the lung and to ensure an adequate space for return flow).

Normal peroral drainage by bechic blast and tussive squeeze is feeble in the aged. Babies have a relatively feeble muscular expulsive mechanism also, and they are further handicapped by the small diameter of their bronchi. The assumption that a small child needs only small bronchi applies only to respiration, not to bronchial obstruction. One millimeter of mucosal swelling all around the wall of the bronchus 4 millimeters in diameter will diminish the lumen to 2 millimeters; but one millimeter all around the wall of a bronchus of 2 millimeters diameter will obliterate it. The smaller the bronchus the more subject its lumen is to obstruction and obliteration. Hence the enormous mortality from bronchopneumonia in infants and young children; all their bronchi are small.

Infections: Acute and chronic, nonspecific and specific, diphtheritic, tuberculous, syphilitic, Vincent's, mycotic and mixed, along with ensuing inflammatory mucosal swelling and inflammatory products, are among the commonest causative factors.[1,2,10,16]

Secretions and Exudates: Fibrinous casts and diphtheritic membranes have long been known as causes of bronchial obstruction. It remained for the bronchoscope to discover that inflammatory purulent and mucopurulent secretions are, primarily, intensely adhesive and cohesive. If allowed to stagnate and thicken into masses they become obstructive *endogenous foreign bodies*, difficult to expel. As shown by the examinations of bronchoscopically aspirated exudates in our clinic there is an increase of fibrinogen concerned in the increase of the cohesiveness and adhesiveness of these exudates. Pathologic bronchial secretions participate in bronchial obstruction in two ways in practically all cases, regardless of the primary cause. (1) They fill in the chinks between the obstruction and the bronchial wall, and (2) they form obstructive masses or plugs by accumulation.[10,16] If this condition is not realized and relief afforded by peroral aspiration *the patient may drown in his own secretions* as detailed in a subsequent paragraph.

The bronchoscopic appearances of obstruction secretions and exudates in the bronchi are shown in color in the published records of the bronchoscopic clinic.[2,10]

Endogenous Foreign Body: In addition to the innumerable *endogenous* foreign bodies in form of accumulations, masses of plugs

of cohesive and adhesive morbid secretions and exudates referred to in the foregoing paragraphs, *crusts* and *sequestra* are common endogenous foreign-body obstructions encountered daily. *Concretions* are rare and true *broncholiths* extremely so. Ther ewere 11 cases of broncholiths bronchoscopically removed in our clinic in a period of 44 years. In the same period 2,076 exogenous foreign bodies were bronchoscopically removed. (See under Impending Drownage of the Patient in His Own Secretion).

Opiates and Atropine act powerfully in two distinct ways to increase the cohesiveness and adhesiveness of secretions and exudates: (a) The desiccating effect of these drugs favors formation of endogenous foreign bodies in form of adhesive masses and crusts.[1-4,9,10] Their thickening effect is largely due to their inhibition of the normal mucosal glandular secretions that would otherwise dilute the inflammatory exudates. The drying power of these drugs is evidenced by the well-known drying of the normal mucosal secretions of the nose, pharynx and nasopharynx. (b) By suppression of the cough reflex, which is the watchdog of the lungs, they annihilate peroral drainage by tussive squeeze and bechic blast.[1-4,9,10] As a result the secretions and exudates accumulate; the longer they accumulate and the greater the accumulation the greater the obstruction. This is further considered in subsequent paragraphs.

Operations: Prolonged suppression of the cough reflex and depression of ciliary action, by *preoperative* and *postoperative medication* with opiates and atropine, is a frequent cause of bronchial obstruction. Attention was first called to the dangers of pulmonary complications from routine use of these drugs in surgical cases by Chevalier Jackson in 1904 and many times in the literature since.[3-5,7,9,10,17] The suppression and stagnation of accumulated pathologic bronchial secretions during prolonged operation under general anesthesia obviously favors the formation of obstructive masses and plugs. Failure to use preoperative and postoperative direct laryngoscopic aspiration of pathologic tracheobronchial secretions is an important factor in some cases. The consequent accumulation of plugs of adhesive and cohesive abnormal secretions became extremely, and often fatally obstructive, especially when bilateral. Unquestionably in a large proportion of the cases the clinical diagnoses of "postoperative pneumonia," in former days, were really atelectasis and not pneumonia, as the internist understands the term.[7,17] In large surgical clinics the surgeon is entirely relieved of anxiety by the anesthesiologist upon whom now rests the responsibility of maintaining a clear channel of air to the alveoli. *Interference with diaphragmatic aid* in tussive peroral drainage by imbalance of relative intrathoracic and ab-

dominal atmospheric pressure during prolonged opening of the abdominal cavity favors stagnation, as does also the patient's reluctance to cough because of the pain it causes. *Postoperative posture* was found to have a bearing on the development of bronchial obstruction. Prolonged dorsal recumbency favors bilateral obstruction, especially in the posterior segments of the lungs. Prolonged lying on the same side favors obstruction of the lobar and segmental bronchi of the lower side. Many cases of "hypostatic pneumonia" were really cases of obstructive atelectasis due to posture.

Impending Drownage of the Patient In His Own Secretions

This pathologic state was first observed in the bronchoscopic clinic in 1903. Many cases due to various causes have been observed since.[2-4,10,22] There are six mechanisms which may operate.

1) Obstruction to expulsion of secretions or exudates.
2) Adhesiveness and cohesiveness or exudates, or massive plugs of exudates or other autogenous "foreign bodies" blocking expulsion of fluid exudates in tracheobronchial lumina of otherwise adequate size.
3) Absence of the cough reflex, or feebleness of the tussive muscular mechanism.
4) Rupture of large abscess, drowning what remains of functioning lung.
5) Perforation of a bronchus by an empyema.
6) Copious flow as in flooding hemorrhage.

1. Obstruction causing the potentially drowning flood may occur in cases of obstruction at the larynx, in the trachea, or by occlusion of both bronchial orifices. The obstruction may be any one of the conditions enumerated under Bronchial Obstruction. In the larynx obstruction may be caused by any proliferative stenotic disease; or by any lesion that interferes with glottic cooperation in the mechanism of cough. This may be by interference with the action of the laryngeal muscles or by fixation of the cricoarytenoid joint. As is well known, the mechanism of cough is as follows: For efficient expulsion by cough a deep breath is taken; the laryngeal lumen is closed, chiefly by the vocal cords; the air pressure in the lungs is increased by thoracic compression; then suddenly the glottis opens coincident with application of additional thoracic compressive effort. Laryngeal obstruction may therefore be in form of local laryngeal lesion or a paralysis due to a central or peripheral nerve lesion. Death of the patient by drowning in his own secretions is a common termination in bilateral *laryngeal paralysis*,[22] contributing as much to asphyxia as the obstruction

to air. It often occurs in the total (complete) laryngeal stage of paralysis when abduction, adduction and "tension" are all gone. In these cases *there is no laryngeal stenosis;* the glottic lumen is ample, but glottic cooperation is totally lacking. The secretion can be seen bubbling upward to higher and still higher levels.

2. The changes in secretions and exudates increasing their adhesiveness and cohesiveness by which their resistance to expulsion by ciliary wafting, tussive squeeze and bechic blast have been stated in preceding paragraphs.

3. Absence or total failure of the cough reflex as a cause of drowning of the patient in his own secretions is more common than is generally realized. (a) It is the terminal phase of a large percentage of deaths from pulmonary disease, as well as in slow deaths from lesions remote from the lungs. The reflexes of the moribund patient become abolished one after another. When abolition reaches the cough reflex, the patient's tracheobronchial tree gradually fills and the patient asphyxiates by drownage in his own secretions when the heart muscle does not get oxygen enough to act. This death by *autodrownage* is manifest by the long known "death rattle." (b) The reflex may be totally abolished by profound central fatigue, or lesions, or toxic conditions. It was first observed bronchoscopically (Jackson, 1911[22]) in a case of acute laryngotracheobronchitis. The introduction of the bronchoscope without anesthesia, general or local, excited no cough.[22] This condition has been observed many times since in this and other toxic diseases.[2-4,7,9,10] Infants have at best a very inefficient cough reflex mechanism; so have the aged. Feebleness of cough reflex and adhesive secretions cooperate in the drowning of the patient in his own secretions. Sedatives, especially opiates with the usual complement of atropine powerfully increase the adhesiveness and cohesiveness of the exudates and at the same time suppress the cough reflex.[2-4,9,10,17]

4. Rupture of a large abscess with impending asphyxia from drownage of the remainder of functioning lung has occurred in our experience. Drownage was averted by prompt bronchoscopic aspiration in a number of cases of bedfast patients in hospitals in which we have maintained a constantly "set up sterile" bronchoscopic room prepared for emergencies.[2,10]

5. Perforation of the lung by an empyema has precipitated impending asphyxia by drownage in a feeble patient in our experience; and asphyxia has been averted by bronchoscopic aspiration as in the cases of rupture of large pulmonary abscess.

6. Flooding hemorrhages may drown the patient in his own blood. This is usually due to opening of a large vessel by (a) external trauma, such as penetrating projectiles or shell fragments;

(b) injudicious traction on a foreign body without prior proper solution of the mechanical problems; (c) erosion of a large vessel by disease involving the tracheobronchial tree such as tuberculosis, aneurysm or malignant disease; (d) in the course of a thoracic surgical operation.

Treatment of Impending Drownage: In the hopeless terminal stage of organic disease, aspiration of the fluids that progressively fill up the tracheobronchial tree is useless. In contrast, however, bronchoscopic (or direct laryngoscopic) aspiration followed by oxygen insufflation in other cases of impending drownage is a life-saving procedure of great efficiency. It is often dramatic in its efficacy.

In cases of impending drownage from inefficient bechic expulsion by patients with a complete bilateral laryngeal paralysis the glottic lumen is ample for respiration, but the patient is cyanotic and extremely dyspneic with a death rattle and a terrifying subjective sense of suffocation. It is marvellous to see the effect of thorough direct laryngoscopic aspiration in these cases. The cyanosis and the rattling of the bubbling secretions in the tree disappear, and the grateful patient, exhausted by day-and-night suffocative anxiety, passes into a deep quiet sleep. The aspiration, of course, does not help the central or peripheral cause of the complete laryngeal paralysis and of course the totally paralyzed cords will never move; but the patient can be prevented from drownage in his own secretions, by bronchoscopic aspiration when secretions and exudates accumulate in his bronchi and lungs. We have had such patients who survived for years, requiring only a few courses of aspiration when an intercurrent infection produced an excessive amount of exudates for a few weeks.

Bronchial Obstruction in Relation to Bronchiectasis

Bronchial obstruction has usually been regarded as the most important causative factor in bronchiectasis, and in the literature "foreign body" (meaning exogenous inspirated foreign body) is always given a prominent place as a cause. Doubtless these opinions are well founded as to obstruction being a factor but the statistics of the Chevalier Jackson Clinic indicate that its importance and the frequence of its occurrence as a cause have been overemphasized. Gross general statistics show that bronchiectasis is a common disease while exogenous inspirated foreign body is exceedingly rare.[14] By means of the bronchoscope a previously unsuspected pathologic feature of bronchiectasis was discovered. Characteristic sputum of bronchiectasis comes up easily and in relatively large quantities. When expectorated in a glass container it usually separates in layers, ordinarily three. As shown by Jack-

son and Jackson[16] this secretion gives no idea of the cohesiveness, adhesiveness and obstructive character of the primary pathologic bronchial secretions. These authors state: Bronchoscopic studies indicate that the two chief, primary causes of bronchiectasis are (1) excessive cohesiveness and adhesiveness of the primary pathological bronchial secretions, and (2) the septic-tank conditions they engender. By the term "bronchiectatic septic tank" is meant not only a receptacle for septic material but a container where the material is changed in physical character and bacterial content by bacterial processes, chiefly saprophytic, in a way parallel to the changes that are utilized by the sanitary engineer to change the physical character and bacterial content of household sewage. The difference is that the concrete walls of the sewage septic-tank are not acted upon by the changed character of the contents whereas the living walls of the bronchi are damaged by the inflammatory changes set up by the irritating character of the by-products of the septic-tank processes. Putrefaction will ultimately change the cohesive, adhesive character of the secretions, so that they can be expelled by cough, but during the delay the septic-tank processes have destroyed the structural integrity of the walls. The chief means of preventing the septic-tank processes is by bronchoscopic aspiration of the pathological bronchial secretions before they have time to stagnate and rot. The primary pus in bronchiectasis is not coughed up; hence when sputum is used, bacterial studies are misleading and autogenous vaccines are inefficient. The important organisms are found only in the residual pus removed bronchoscopically from primary foci after the bronchiectatic septic tank has been emptied by bronchoscopic aspiration. For removal of this diagnostically (and therapeutically) important specimen of residual pus, a small special specimen collector is invaluable. The collector is attached to the aspirating tube. The aspirations should be done by the synergetic technic in which full advantage is taken of the tussive squeeze to bring the pus up from the lobar periphery.[1] These aspirations take the load off the cilia. In fully developed bronchiectasis these aspirations are, of course only palliative. It has been abundantly demonstrated that, used early enough, they are prophylactic. The bronchoscopic appearance in the bronchiectatic septic tank are shown in color in publications of the bronchoscopic clinic.[2,10]

Prophylaxis of Bronchiectasis: Abundant experience of the bronchoscopic clinic shows that bronchiectasis is to a great extent a preventable disease.[2,10,17] We believe it is safe to say that the incidence of bronchiectasis could be reduced to relative rarity if every patient with tardy recovery from acute infections of the lower respiratory tract were promptly given the benefit of bronchoscopic

aspiration. So-called "unresolved pneumonia," residual interstitial pneumonitis, and other pathologic conditions may be placed in the same category. Bronchoscopy, in addition to its prophylactic value, often demonstrates that the pathologic conditions present were not pneumonic in origin. Unquestionably many forms of pneumonia, virus pneumonia for example, are primarily diseases of the bronchial system with atelectasis, emphysema, interstitial pneumonitis, and other secondary changes in the lungs. Infective disease of the nasal cavities and nasal accessory sinuses must not be neglected. In addition prompt bronchoscopic arrest of the lingering remainders of acute infections of the lower respiratory tract, causing obstruction of one or more branch bronchial orifices, is absolutely essential. The thick, tenacious secretions, occurring as localized obstructions, with which peroral pulmonary drainage is unable to cope, must be removed by synergetic bronchoscopic aspiration. Along with this must go a strict *conservative regimen* to maintain maximum resistance. This regimen should include (1) physical rest, (2) good ventilation, (3) sunshine, (4) regulation of diet, (5) maximum vitamin intake constantly maintained, (6) any chemotherapy or other medication that may be indicated, (7) prohibition of all sedatives and antibechics. Unless the regimen is strictly enforced the patient will use up increased energy in greater activity instead of utilizing it to build up a reserve of resistance. If this plan of bronchoscopic aspiration combined with a strictly enforced regimen of medical care and management were followed after every acute infection of the tracheobronchial tree that shows a tendency to linger, bronchiectasis would become a rare disease. We have abundant clinical records affording documentary evidence of this clinical fact.

Bronchial Obstruction in Pulmonary Abscess

The bronchoscopic appearances vary in the different types of pulmonary abscess. In *embolic abscess* there is, in most cases, no obstruction other than exudates in the larger bronchi. The mucosa is usually reddish and adherent patches of yellowish or brownish purulent secretion are usually visible. When the segmental orifices are reached by seriatim inspection, some of them will usually be found to be swollen partly or completely shut. Pus is usually present in some of the segmental orifices. After it is aspirated so as to give a clear field pus will usually be seen to emerge from some of the branch bronchial orifices at each tussive squeeze. In case of small multiple abscess, careful watch of each small segmental branch orifice at repeated bronchoscopies may be necessary as the amount of pus may be small in any one branch. An unpleasant odor may be noted coming through the bronchoscope but it is not

usually found as in some types of abscess and may be absent. Odors are often noted coming through the bronchoscope when not noticeable on the breath or sputum. In the less common type in which an embolic abscess ruptures into a larger bronchus, sloughs and grayish or greenish debris along with purulent and bloody or brownish material and clots may be found. After this is aspirated there is usually little obstruction to drainage, especially if peroral aspirations are repeated regularly. If bronchoscopic aspiration is omitted stagnation and obstruction may result in secondary abscess formation and massive obstruction. With frequently repeated aspirations small granulations are seen to form around the orifice; but good drainage can be maintained, especially if there are no other foci, and systemic sepsis can be controlled by chemotherapy and the other usual means. In time, if resolution does not take place, and external surgery is not done, the bronchoscopic appearances become those of chronic abscess. These appearances have been published in color.[2,10]

Putrid Pulmonary Abscess, if large and of fulminating type, usually creates demand for bronchoscopy to relieve dyspnea or to prevent asphyxia. In such cases the bronchoscope usually encounters constant bleeding, which requires the aspirating bronchoscope to maintain a clear field. Foul brownish obstructive material is usually found in both main bronchi, even in cases in which a single abscess is discharging into one of the smaller bronchi of one lung. After synergetic bronchoscopic aspiration of this stagnant putrid obstructive bronchial contents, constant drainage is maintained through the drainage canal of the aspirating bronchoscope, so that the source of the obstructive material can be located. When found, the entrance to the suppurative focus will usually appear relatively small and almost or entirely closed by swollen mucosa and if more than a week or two has elapsed the orifice will have a fistulous appearance, and it will be obstructed by exuberant granulation tissue. As a rule it is not necessary to insert a small bronchoscope or aspirating tube into an abscess cavity. If it is decided to postpone decision as to external operation, drainage by eynergetic aspiration without anesthesia general or local will be quite efficient and the fistulous orifice can be maintained patulous during the "conservative regimen" period of observation.

Posttonsillectomic Pulmonary Abscess: There is much clinical evidence to show that posttonsillectomic abscess is usually embolic in origin.[2,6,10,13] Be this as it may the bronchoscopic appearances are the same as are found in embolic abscess as just described, and immediately instituted bronchoscopic aspirations yielded a

large percentage of cures even before the discovery of penicillin.[13] With penicillin or other chemotherapy the percentage is higher. In our clinic, however, decision as to plan of treatment in the particular case is reached at group conference as hereinafter mentioned.

Pulmonary Abscess Due to Exogenous Inspirated Foreign Body: Typical pulmonary abscess showing a fluid level in the roentgenogram is exceedingly rare as the result of exogenous inspirated foreign body. This fact has been fully established by the published records of the bronchoscopic clinic.[2,10,11,13,15] *Drowned lung*, with the part of the bronchial tree distal to the obstruction caused by the foreign body and the reaction of the bronchial tree to its presence filled with pus is not uncommon; but disintegration of pulmonary tissue and the development of a cavity with an abscess wall is exceedingly rare. This statement is based on records of exogenous inspirated foreign bodies of sojourns in a bronchus for periods ranging up to 50 years. The pathologic process developed in the reaction to the presence of a foreign body are those of building up a barrier against invasion rather than disintegration of tissue such as is commonly seen in pulmonary abscess. The so-called "bronchiectatic abscess" has been observed in a few cases, but bronchiectasis itself is not a common sequela of exogenous inspirated foreign body, as mentioned in a previous paragraph. Suppurative processes engendered by prolonged sojourn of such foreign bodies is followed by complete recovery in most cases. In a relatively small percentage of such cases, synergetic peroral aspirations to take the load off the cilia for a time are useful, and in an occasional case surgical measures may be required.[2,10,11,13,15]

Chronic Pulmonary Abscess: The bronchoscopic appearances in cases of chronic pulmonary abscess always show that it is essentially an obstructive disease. The characteristic bronchoscopic picture is one of pus endeavoring to escape through an inadequate aperture. After preliminary aspiration of exudative material accumulated in the bronchial lumen, pus is seen to emerge from fistulous or pathologic bronchial orifices synchronously with the tussive squeeze of each cough. After good drainage by synergetic bronchoscopic aspiration has cleared the field the character of the obstruction is easily and satisfactorily studied. The most common forms of obstruction are (a) thick cohesive and adhesive semisolid exudates; (b) plugs; (c) crusts; (d) granulations and granulomas; (e) firm nodules of more or less epithelialized tissue; (f) cicatricially contracted orifices; (g) infiltrated and thickened chronically inflamed mucosa; (h) debris, sometimes containing cartilaginous sequestra.[2,10]

Bronchoscopic Appearance of Tuberculosis

From the purely pictorial viewpoint bronchoscopic images of endobronchial tuberculous lesions are the most varied and interesting in the entire field of peroral endoscopy. There is nothing approximating the degree of standardization seen in the histologic characteristics of tuberculosis. In addition to the already published illustrations[10] there are over 80 sketches in color remaining in our collection. Although the changes in the appearances develop slowly they never cease changing, and the changes often affect one part of the image more than another. Progressive changes in form are slower than changes in color. The most frequently observed elements in the bronchoscopic image are (a) chronic tracheitis which is probably not tuberculous, with some branching vessels faintly visible. (b) Diffuse inflammatory mucosa, with color slightly redder than normal and with a surface on which a slightly roughened or granular condition can be seen by the oblique illumination of the distal light. This appearance may be limited to a small area, but often it is varied by small or large but low nodules representing a number of later stages of the development toward a surface of one or many nodules large enough to be plainly visible. (c) Small mounds or nodules whose surface may be smooth and glistening, but more often is rather dull with the appearance of chronic mucosal inflammation; this lesion if it is the only one and if no excess of secretion is present, is not obstructive enough to produce a wheeze audible at the proximal tube mouth. (d) One or many nodules are visible and they are obstructive enough to cause a wheeze audible at the tube mouth but there is no erosion of the surface; (e) nodules usually somewhat irregular in size and outline, and encroaching on the lumen sufficiently to cause a wheeze plainly audible as the secretions are seen to bubble back and forth in the narrowed lumen, part or all of which may be nodular; (f) nodules closing the lumen at the start of expiration, opening at maximum inspiration, producing obstructive emphysema by the trapped air (ulceration may be developed in this stage). (h) Complete occlusion of lumen so that no air can get in or out (no wheeze is audible, and atelectasis is found over the tributary area). Sometimes in case of tuberculous abscess the valvular mechanisms are seen to operate at the orifice of the abscess. (i) Granulations, granulomas, tuberculomas and ulcerative areas are visible in various numbers, sizes, and positions. (j) Scar tissue or a subepithelial fibrosis may be detectable in various forms and areas; they are usually paler in color and associated with stenosis; they may appear as concentric or excentric strictures varying in lumen from a slight diminution to a tiny pinhole or complete occlusion.

The foregoing descriptions are simplified for sake of clarity. They may be modified by various factors such as angle of the axis of the diseased bronchus relative to the bronchoscopic axis; location of the lesion relative to bronchial wall or branch bronchial orifice; secretions, crusts, caseous material, blood staining, blood clots, intrusion of debris from peribronchial nodes, silicotic material, anthracotic material and all sorts of combinations of the pictorial images included in the foregoing enumerations of elemental components. The possibility of a malignant implantation on a tuberculous lesion must always be borne in mind.

Adenopathy: Lymph nodes, singly or collected in masses, are frequently observed as causes of bronchial obstruction. The intrusion of a suppurating node has long been known as an obstructive lesion at necropsy. At bronchoscopy they are usually encountered as (a) masses of accumulated debris in a bronchus or as (b) a compressive obstruction without intrusion. The latter type of lesion almost always produces a valvular type of obstruction, regardless of the particular character of the adenopathic, pathologic, process, tuberculous, syphilitic, mycotic, malignant, or secondary to a suppurative focus.[2,7,8,12]

Bronchial Obstruction by Tumor

At bronchoscopic examination the obstruction produced by a tumor may be by (a) an endobronchial mass; (b) compression stenosis of a peribronchial tumor; or (c) compression stenosis by lymph nodes. Two or all of these types may coexist in the same patient at the time of the first or any subsequent examination.

Benign Bronchial Tumors are characteristically obstructive lesions. All types of valvular obstruction are each present at some stage of the tumor's development.[7,8]

Bronchoscopic Appearances of Benign Tumors: Any endoscopic image is made up of form and color. Form concerns obstruction as well as size. In *form* benign tumors appear as (a) a sessile mounding of the wall into the lumen; (b) an irregular globular tumor filling the lumen, more or less completely; (c) pyriform or pedunculated tumor, pedicle visible or invisible until exposed by bronchoscopic manipulation of the tumor; (d) cauliflower-like fungations. Rhythmic enlargement and elongation of lumen followed by diminution and shortening affect form greatly in soft tumors such as multiple papillomata and fungating granulomata. Ulcerogranulomata are affected less. The form of tumors made up largely of inflammatory hyperplasia, change little; fibroma very little. Luminal enlargement and diminution may be accompanied by flopping of a pedunculated tumor. Luminal movements are greater, usually, in case of benign as compared to malignant

bronchial tumors. Except papillomata and granulomata, benign tumors seldom involve the entire periphery, hence luminal movements are seldom concentric. Another morphologic difference is that benign tumors, probably because of long slow growth and lack of mural invasion, are often caged in a bronchial dilatation they have made for themselves.[2,7,8] This type of dilatation is *not* bronchiectatic and it is *not* distal to the tumor. It is probably due to years of bulging pressure on the bronchial wall, 20 or more times a minute. The duration of malignant tumors is too short for the formation of a bulge in the bronchial wall. Except for this bulging cage, which tends to form an upper and a lower valve seat, the valvular mechanism is the same as that of malignant tumor. The double valve seat favors development of *alternating obstructive atelectasis and emphysema* (Jackson, 1917[23]).

The *color* of benign tumors varies. The most common are as follows: (a) whiter than the neighborly mucosa in case of fibromata and some papillomata; (b) yellowish if largely lipomatous; (c) pink in case of adenoma, granuloma and most papillomata; (d) irregular patches of slightly roughened eroded surface; (e) branching vessels; (f) bleeding points; (g) diffuse blood filmed surface; (h) patches of adherent secretion obscuring more or less of the tumor.[2-8,10,15,20,21] (Typical bronchoscopic appearance of tumors are illustrated in color in references[2,10,23]).

There is an impression of benignancy made upon the experienced bronchologist on looking through the tube at a benign growth that is difficult to describe in words, but which probably all bronchologists have felt. "Looseness" does not accurately describe it; neither does "Lack of the firmly rooted fixity of a malignant tumor," though these words suggest it. It is not mentioned here as of value diagnostically, but rather as related to a description of bronchoscopic appearance.

Bronchial Obstruction by Malignant Tumor: Neoplastic malignant bronchial obstruction occurs in three forms, compressive, annular and sessile. The first stage of obstruction by carcinoma and by sarcoma is of the by-pass type, permitting ingress and egress of air, though in diminished volume. It is practically always accompanied by wheezing heard at the open mouth. At a later stage the obstruction increases and develops the check-valve type, producing first emphysema then atelectasis; sometimes *alternating atelectasis and emphysema*, and in some cases *concurrent emphysema and atelectasis* in respective lobes or segments. Later it develops the stop-valve type in which both ingress and egress of air are completely obstructed. The *bronchoscopic appearances* at these various stages have been described and illustrated in color.[7,10,12,17] Compression stenosis by a peribronchial malignant

tumor follows the same sequence of by-pass valve with wheeze, check valve with emphysema or atelectasis or both concurrently and finally stop valve. In the earlier stages of neoplastic bronchial obstruction two types of mechanism are observed, the expansile and the flapper mechanisms. Both differ from similar mechanisms as seen in two forms, the annular and the sessile. In the annular mechanism the entire circumference being involved, the lumen is seen to enlarge and diminish concentrically. In the sessile form, the tumor mounding inward from one side leaves a crescent chink on inspiration; the crescent disappears or diminishes on expiration. The flapper mechanism is also different in appearance as compared to the appearance of flapper mechanism of benign tumors, particularly the benign growths that are capsulated and pedunculated. Flapper tissue in malignant bronchial growths is usually in form of fungations, without a capsule. Ultimately all check-valve mechanisms in malignant bronchial disease end in stop-valve obstruction which means total occlusion. The obstruction is so great that the inspiratory expansile movement cannot create a lumen. As the neoplastic infiltration increases a characteristic *fixity* is readily recognized by the eye familiar with the normal flexibility and movements of the tracheobronchial tree. The rhythmic enlargement of lumen on inspiration is replaced by what might be called a "wooden" appearance.

In the early stages the surface of a bronchial carcinoma is usually slightly nodular or bossellated, but the color differs noticeably from that of the normal mucosa. If pale, branching vessels may be apparent, but when the color is more reddish, vessels are invisible. Within a few months the endoscopic appearances become more conspicuously nodular. The surface is seen to be exfoliating; erosion and ulceration soon follow but an opening of a lumen does not usually follow. Fungations develop and fill the bronchus with anexuberant mass covered with a blood stained purulent surface. This is the stage of stop-valve obstruction. Any picture of malignant neoplastic bronchial obstruction at the main bronchial orifices may be modified by the *broadened carina* that indicates adenopathy, though the adenopathy may not be primarily malignant. It is extremely rare to find a bronchial carcinoma or sarcoma that is pedunculated in form, but exuberant fungations or nodules may lead to a false impression of such a form. Another rarity is to find the endoscopic picture modified by cicatricial stenosis which presumably antedated the neoplastic process. The same may be said of exogenous foreign body. Carcinoma and sarcoma both occur as implants on tuberculous lesions. These rare forms of lesions are each represented in our collection of sketches of bronchial obstructions.

Prophylaxis of Bronchial Obstruction

Obviously, some forms of bronchial obstruction are inevitable, tumors for example; on the other hand, many kinds of bronchial obstruction are preventable, as is evident from the list of etiologic factors enumerated in a preceding paragraph.

Great progress in prophylaxis of bronchial obstruction has been made since the more general recognition of the century old therapeutic error in the use of opiates to suppress cough. It is over 30 years since attention was first called to this error by one of us,[3] and much has been written since,[2,4,10] but it is only in recent years that the error has found a place in general medical literature. Iteration and reiteration will be necessary to eradicate the error of the drugging to sleep of the cough reflex which is the watchdog of the lungs.[3-6,10,13,17] It is pathetic to think of one of the greatest internists of the twentieth century dying of acute infective inflammatory bronchial obstruction praising the blessing of opiates in suppression of his cough.

It is unnecessary here to call attention to the fact that the perfection of the means of prevention of operative and postoperative bronchial obstruction has had a large share in the marvellous reduction of postoperative complications and operative mortality of general as well as thoracic surgery. Particularly advantageous has been the development of anesthesiology as a specialty and the placing on the shoulders of the anesthesiologist the responsibility of maintaining before, during and after operation a clear efficient gaseous channel to the alveoli. Most of this work can be done with the silk-woven aspirating tube introduced into the trachea through the laryngoscope and passed down to the carinal level, with the patient in any position, even prone for laminectomy.

Treatment of Bronchial Obstruction

One glance at the list of causes of bronchial obstruction would render obvious the impossibility of proper consideration here of even the bronchoscopic phases of treatment of such a variety of lesions. A few broad generalities may be stated.

The bronchoscope has removed the treatment of bronchial obstruction from the domain of theory and inference and has placed it on a plane with other departments of medical science in which therapy is based on direct inspection of pathologic tissue changes, laboratory examination of exudates and histologic examination of pathologic tissue. Confusing, often misleading examinations of sputum, with its abundant contaminations from teeth, tonsils and sinuses, have been replaced by microscopic, cytologic and cultural examination of uncontaminated specimens obtained by

aspirator and sterile cotton sponge direct from the focus of disease. Bronchoscopic biopsy and accurate localization of tumoral and other lesions give the surgeon invaluable information for planning operative procedures. Bronchoscopy, therefore, places treatment of bronchial obstruction on the scientific basis of dealing with pathologic conditions known to be present and accurately located in the particular case. When, however, it comes to the bronchoscope as a means of treatment, it may be said that in the treatment of exogenous inspirated foreign body, the close to 100 per cent of successful removals give it a unique position. In all other forms of bronchial obstruction its relations to treatment are those of a speculum, through which treatment may or may not be indicated, according to the pathologic conditions present in the particular case. In a broad and general way it may be stated that, apart from diagnosis, the chief therapeutic duty of the bronchoscope is to establish a clear and adequate airway to the alveoli and to reestablish *normal physiologic upward peroral drainage* by ciliary wafting, tussive squeeze and bechic blast. No matter what else is done, the patient will never have healthy lungs as long as stagnation persists. Prevention of stagnation by a series of systematic peroral aspirations of the tracheobronchial tree by synergetic aspirations with the silk-woven aspirating tube, after postoperative convalescence is well established, will greatly aid restoration of peroral drainage by taking the load off the clogged cilia, and may possibly promote development of new cilia. Not only after pulmonary resections, but also after external drainage of empyema, bronchoscopic removal of bronchial obstructions and a series of peroral aspirations may be of great aid in the establishment of peroral drainage. Such aspirations, when indicated, are easily done by the anesthesiologist, on whom the maintenance of a clear airway largely depends.

For many years it has been our custom to submit the case of the particular patient for free discussion at the weekly Chest Conference. In this conference participate the internist (or pediatrician), phthisiologist, thoracic surgeon, radiologist, bacteriologist, pathologist, and bronchologist. If surgical treatment is postponed, synergetic bronchoscopic aspirations, and other indicated bronchoscopic therapy, are added to general medical care and management, pending further discussion at a subsequent conference.[19]

SUMMARY

1) Bronchial obstruction, temporary or prolonged, is a universal pathologic occurrence; it is common among the newborn; it affects everyone many times later in life, and it is commonly the terminal phase in slow deaths from any cause. Only the cough reflex pre-

vents us all from drowning in our own secretions. Only the cough reflex prevents extermination of the human race by bronchial obstruction.

2) The power of bronchial obstruction to cause bronchial and pulmonary disease is threefold. It acts as a primary, a predisposing and a perpetuating factor.

3) An effort is made to clarify the confusion in the literature due to theory and inference regarding valvular obstruction. Complete obstruction of a bronchus, as shown at necropsy, has been well known since the days of Hippocrates and Galen. The bronchoscope, 26 years ago, revealed the clinical fact that in the living bronchi, normal rhythmic respiratory movements produced valvular types of obstruction that caused emphysema and atelectasis in the respective tributary areas. The mechanism of two of the types was similar to the stop valves and check valves common in mechanical engineering, but the most frequently encountered type of mechanism was unknown to mechanical engineers. This newly discovered mechanism was named the "expansile check-valve." It converts the rhythmic respiratory to-and-fro flow of the gaseous contents of the bronchi into a one-way flow. The inspiratory diametric luminal enlargement opens a chink past an obstruction, but the expiratory diametric luminal diminution closes the chink at the beginning of expiration, trapping the air before it can escape. Though the quantity trapped at each expiration is small, repetition 18 or more times a minute soon results in emphysema of the tributary lung, lobe or segment. This expansile type of one-way valve is irreversible. The two types of one-way valvular mechanism known to mechanical engineers, the ball valve and the flapper valve, are reversible; and when they occur in reversed position in a bronchus, they cause atelectasis rapidly. They are often seen to produce atelectasis in a few minutes, whereas absorption of air by the circulation after a stop-valve obstruction usually takes 24 hours or more to cause atelectasis. A flapper valve may cause atelectasis in one lobe and at the same time an emphysema in another lobe.

4) The expansile check-valve mechanism is seen at some stage in practically every disease of the lung. It occurs not only in endobronchial conditions, but also in neoplastic, adenopathic and other compression stenoses.

5) Various causes of bronchial obstruction are discussed. The most frequent are pathologic secretions and exudates that the cough reflex cannot expel because of their greatly increased adhesiveness and cohesiveness. Difficulty of expulsion is also greatly increased by accumulation. Opportunity for accumulation occurs when the cough reflex is fatigued, feeble, inefficient from lack of

glottic cooperation, or suppressed by toxemia, or alcohol, or drugs, especially opiates and other sedatives. Opiates are the most frequently prescribed frustraters of the vital defense of the lungs. Their routine use as antitussives is one of the most deplorable and widely spread therapeutic errors in the history of medicine.

6) It was discovered bronchoscopically many years ago that opiates and atropine act powerfully in four different ways in promoting bronchial obstruction. (a) The dessicating effect of these drugs greatly increases the adhesiveness and cohesiveness of pathologic secretions and exudates, and this change enormously increases the difficulty of expulsion by nature's defensive mechanism, ciliary wafting, tussive squeeze and bechic blast. (b) The dessication also favors coagulation into firm plugs. (c) By suppression of the cough reflex opiates give time for accumulation, coagulation and plug formation. (d) By suppressing cough, opiates directly cripple the natural machinery of defense of the lungs. They drug the watchdog to sleep. It is hard to conceive of a drug better adapted to the promotion of bronchial obstruction.

7) Peroral synergetic aspiration of the bronchial tree is an important means of prophylaxis. It is easily done with a silk-woven aspirating tube inserted through the laryngoscope, or with a bronchoscope inserted without the laryngoscope. Deep insertion of an aspirating tube is unnecessary; the tussive squeeze will force exudates up into the large bronchi. Chronic bronchitis and bronchiectasis are largely preventable diseases. Prompt arrest of beginning stagnation of pathologic secretions and exudates, when subacute bronchitis lingers after acute infections of the respiratory tract, will prevent chronic bronchitis and bronchiectasis which are so often the sequelae of such acute infections.

8) By diagnostic bronchoscopy the treatment of bronchial obstruction has been removed from the domain of theory and inference and placed upon a plane with other departments of medicine and surgery in which therapy is based on direct inspection of pathologic tissue changes, laboratory examination of exudates and histologic examination of pathologic tissue specimens. As a means of treatment its close to 100 per cent of successful removals of exogenous foreign bodies gives bronchoscopy a unique position; but exogenous foreign body as a cause of bronchial obstruction is relatively rare. In all other forms of bronchial obstruction the relations of the bronchoscope to treatment are those of a speculum through which treatment may or may not be indicated, according to the pathologic conditions found in the particular case. Two important duties of the bronchoscope in all kinds of cases, are to maintain constantly a clear and adequate airway to the alveoli, and to reestablish normal physiologic peroral drainage by ciliary

wafting, tussive squeeze and bechic blast. No matter what else is done in the way of operative or non-operative treatment, the patient will never have healthy lungs as long as stagnation persists. Prevention of stagnation by a series of systematic peroral aspirations of the tracheobronchial tree, as soon as convalescence is established will greatly aid in restoration of normal peroral drainage, by taking the load off the clogged cilia. After operations such as pulmonary resections, or external drainage of empyema, bronchoscopic removal of obstructive bronchial exudates and a series of peroral aspirations are usually of great aid in reestablishment of normal physiologic drainage of the lungs. Such aspirations are easily done by the anesthesiologist, on whom maintenance of a clear airway so largely depends.

9) In case of a patient with impending drownage in his own secretions, resuscitation by bronchoscopic aspiration is dramatic.

SUMARIO

1) La obstrucción bronquial, temporaria o prolongada, es una entidad Patológica, que courre universalmente; es común entre los recién nacidos; afecta a todos, muchas veces durante la vida, y es comunente la fase terminal de la muerte lenta, por cualquier causa. El reflejó tusígeno nos proteje de morir ahogados en nuestros propias secresiones. Solamente el reflejo tusígeno previene la esterminación de la raza humana por la obstrucción bronquial.

2) La obstrucción bronquial afecta los bronquios y los pulmones en tres maneras, a saber: en forma primaria, por predisposición y en forma permanente.

3) Se hace en esfuerzo para aclarar la confusión en la literatura, debido a la teoria de la obstrucción valvular. La obstrucción complete del bronquio, como se ve en la autopsia, es bien conocida desde los tiempos de Hipócrates y Galeno. Hace 26 años, el broncoscopio, demonstró el factor clínico en una persona viva, que los movimientos respiratorios, rítmicos, normales, producen una obstrucción de tipo valvular, que a su vez, produce enfisema y atelectasia en las zonas correspondientes. El mecanismo de dos, de los valvulas es similar, a aquellos, que se usan en ingenieria mecánica, pero, la que se encuentra mas frecuentemente no es conocida en ingenieria mecánica. Este nuevo mecanismo, se lo llamó, "La valvula expansible de control." Convirte los dos corrientes del ritmo respiratorio de los gases bronquiales, en una. La dilatación del bronquio en la inspiración, abre una endidura después de la obstrucción, pero cuando el bronquio se contrae, en la expiración, cierra la endidura al comienzo de esta y atrapa el aire antes que pueda salir. Se bien es cierto, que la cantidad retenida en cada expiración, es pequeña, cuando esta se repita 18 o mas veces por

minuto, infaliblemente, el enfisema no tardará en instalarse en las regiones afectados. Este tipo de válvula es irreversible. El mecanismo de los dos tipos de válvulas simple, conocidas en engeniería mecánica, la "válvula a bola" y la "valvula de aleteo," son reversibles; y cuando esto ocurre en posición inversa en un bronquio, produce la atelectasia rápidamente. Estos producen atelectasia en pocos minutos; en cambio, en el caso de la válvula de cierre la obstrución se produce en 5 o 6 horas. La válvula "de aleteo" puede causar atelectasia en un lóbulo y al mismo tiempo enfisema en otro lóbulo.

4) El fenómeno de la válvula de suguridad expansible, se observa en diferentes grados, en casi todas las enfermedades del pulmón. Se presenta no solamente, en afecciones endobronquiales, sino también en neoplasmas, adenopatias y otras estenosis por compresión.

5) Se han discutido varias causas de obstrucción bronquial. Las mas frecuentes son las secreciones patológicas y exudados, que el reflejo tusígeno no puede expeler, debido a la coherencia y adherencia de los mismos. La expulsión es todavía mas deficil cuando esta se acumula. La acumulación se produce especialmente cuando el reflejo tusígeno está fatigado y débil; dificultado por falta de cooperación de la glotis; disminuido por la toxemia, alcoholismo, drogas, especialmente opiáceos y otras sedantes. Opiáceos son los mas frecuentemente usados para frustrar la defensa vital de los pulmones. Su uso como antiemético, es el mas deplorable y común de las herrores terapéuticos de la historia de la medicina.

6) Ha sido descubierto hace muchos años, por la broncoscopia, el hecho que los opiáceos y la atropina, favorecen la obstrucción bronquial en cuatro maneras diferentes: (a) El efecto desecativo de estas drogas aumenta grandemente la coherencia y adherencia de las secreciones patológicas y exudados; y esto naturalmente aumenta la dificultad de expulsarlos por los mecanismos de defensa naturales, por la ondulación de las cilias, contracción de la tos, etc.; (b) La desecación también favorece por coagulación, la formación de tapones duros; (c) Los opiaceos desminuyen el reflejo tusígeno, el cual produce la acumulación, coagulación y la formación de tapones; (d) Al desminuir el reflejo tusígeno, los opiáceos, comprometen el mecanismo de defensa natural de los pulmones. Ellos narcotisan el "perro guardian." Es deficil de concevir una droga que mejor se adapte al caso de promover la obstrucción bronquial.

7) La aspiración del árbol bronquial es muy importante desde el punto de vista profiláctico. Se hace facilmente con un tubo tejido de seda, el cual se inserta con la ayuda del laringoscopio o con el broncoscopio, el cual se inserta atravez del laringoscopio.

No es necesario, la inserción del tubo, muy profundamente. La fuerza de la tos, expelerá la secreciones en los grandes bronquios. La bronquitis crónica y la bronquiectasia son enfermedades prevenibles. Al evitar el principio del estancamento de los secreciones patológicas y exudados, cuando una bronquitis sub-aguda se prolonga, después de un proceso respiratorio agudo, se prevendrá la bronquitis crónica y la bronquiectasia, los cuales son frecuentes complicaciones.

8) Gracias al diagnóstico broncoscópico, el tratamiento de la obstrucción bronquial se ha transformado, desde un campo teórico, en el mismo plano con los otros departamentos de medicina y cirujía, en los cuales el tratamiento esta basado en inspección directa de las lesiones patológicas, exámenes de lavoratorio de los exudados y exámenes histo-patológicos de las lesiones. Como tratamiento, para extraer cuerpos extraños, con casi 100 por ciento de éxito, coloca la broncoscopía en una posición única. Sin embargo obstrucción bronquial causada por cuerpos extraños es relativamente rara. En todas las formas de obstrucción bronquial, el broncoscopio está relacionado con el tratamiento, en la misma forma que el espéculo, a travez del cual se aplica el tratamiento indicado, de acuerdo con el caso. Dos condiciones son importantes en broncoscopía, en todos los casos, ellos son, el mantener abiertos constantemente las vias aereas con los alveolos y reestablecer el drenaje fisiológico por boca, por medio de las cilias bronquiales, contracción de la tos, etc. El enfermo no mejorará, a pesar del tratamiento quirúrgico o no quirúrgico, si el estancamiento bronquial persiste. El estancamiento se puede prevenir con una serie de aspiraciones del árbol bronquial, tan pronto como la convalascencia se ha instalado, ayudará a establecer el drenaje oral y aliviará el funcionamiento de las cilias bronquiales. Después de operaciones, tales como resecciones pulmonares, o drenaje externo de un empiema, la aspiración broncoscópica de las secreciones bronquiales y una serie de aspiraciones orales, son una gran ayuda para establecer un drenaje fisiológico de los pulmones. Tales aspiraciones son realizados facilmente por el anestesista, el cual es responsible en mantener las vias aereas abiertas.

9) Se obtiene un resultado mágico con la aspiración broncoscópica, en el caso de un enfermo que se esta ahogando en sus propios secreciones.

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Pulmonary Disease, Associated with Cystic Fibrosis of the Pancreas*

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Chronic Pulmonary Infection, associated with Cystic Fibrosis of the Pancreas, has been recognized as a clinical entity for over a decade. Although the respiratory symptoms and signs are not always the initial ones, they are usually the most startling part of the picture, and often the ones for which the patient is first brought to the physician. Until the last few years, the appearance of the pulmonary disease in this condition made the prognosis grave, and most of the patients were lost. Our increased knowledge of the disease over the last few years, and our new ability at early recognition, has permitted us to institute vigorous treatment before irreversible changes in the pulmonary system have taken place. We are now able to save many patients, and in some to give supportive treatment until the time when they may lead relatively normal lives.

As the symptoms usually develop in the first year of life, the patient is seen first by the general practitioner or the pediatrician, and by the chest specialist usually only in consultation. There are, however, a few patients seeming to have the disease in a milder form, who live to the later years of childhood without having the true condition recognized. In any child patient with a chronic respiratory infection, and especially if it extends from the tip of his nose to his alveoli, with sputum and a negative tuberculin, this disease should be thought of at once. In the infant patients of a decade ago, the diagnosis was often made only at necropsy by a section of the pancreas. But, if suspected at all, the diagnosis can be made during life at all ages, usually, if a careful history be taken, with special reference to feeding difficulties in early infancy, and may be confirmed in most cases by laboratory analyses, the most important of which is an examination of the duodenal contents.

Case 1: N.P., female, age 27 months, entered with complaint of persistent cough for 26 months, and rectal prolapse for one year. One male sibling was normal. The cough, beginning at age of one month, resembled pertussis in its onset. She developed purulent rhinitis, otitis

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media, and bronchopneumonia at three months. Since then she has had poor appetite, has been underweight, cough has persisted and she has had continued respiratory infections, with bronchopneumonia demonstrated several times roentgenographically. Measles was contracted from the brother at ten months and modified by placental extract. Examination showed a thin child weighing 21 pounds, with a cyanotic tinge to skin and lips, breathing was rapid and labored. The thorax showed marked pigeon-breast with bending at the costochondral junctions. The anterior fontanelle was open. There were scattered coarse rales, inspiratory and expiratory, throughout the lung fields. Mucopus was present in the pharynx and on the turbinates. The white blood count was 19,720, with 86 per cent polymorphonuclears, HB 91 per cent S., red blood cells 5,110,000. The urine showed a one plus sugar on several occasions. There was a marked increase in the amount of fat in the stools, and occult blood was present. Wassermann, tuberculin and coccidioidin tests were negative. Roentgen studies showed the antra and ethmoids to be filled with material of soft tissue density, as were the cells of the left mastoid; of the chest, there was an emphysematous condition of the lung fields with scattered densities (Figs. 1 and 2). Skin sensitization tests were all negative. Intranasal oxygen and sulfathiazol were started, but the course was rapidly down hill, and increasing cyanosis occurred. The abdomen became markedly distended, acute dilatation of the stomach was temporarily relieved, but the child died six days after admission.

Necropsy report: The lungs were similar in size and appearance. The left lung weighed 110 grams and the right 100 grams. The external surfaces were smooth, pale and pink. The lower lobes were slightly firmer than the upper. The cut surfaces were pale, tan-pink and studded with dilated thin-walled bronchi in the peripheral zones, from which thick yellowish-green pus exuded. Histologically, there were many dilated bronchi with inflamed thickened walls containing polymorphonuclear exudate. No epithelial metaplasia was found. Scattered groups of alveoli

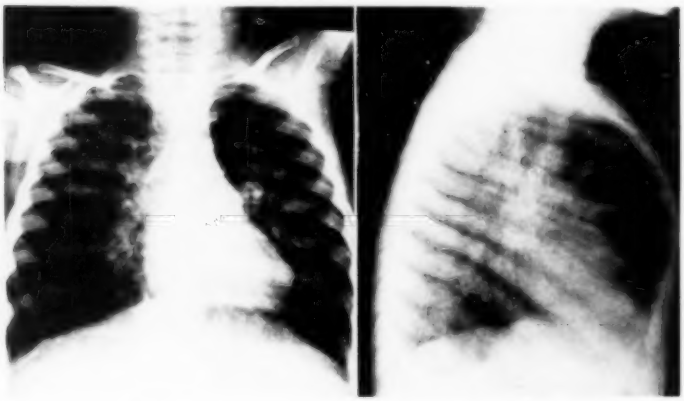

FIGURE 1 FIGURE 2

Fig. 1. Case 1: Scattered densities in lung fields, with emphysema.
Fig. 2. Case 1: Lateral view of chest.

were filled with autolyzing leucocytic exudate or blood. There were patches of dilated and ruptured alveoli. Purulent exudate filled the larger bronchi and trachea. The hilar lymph nodes were large and moderately firm, grayish-white and translucent. The liver weighed 400 grams. The cut surface was yellowish-tan and glistening. Histologically, many of the parenchymal cells contained fat deposits. The hepatic and common bile ducts were patent. The gall bladder was very small and atretic, consisting of only a fibrous tissue without visible lumen. The cystic duct was a thin, tortuous, fibrous cord. The pancreas was firm and cut with increased resistance. Histologically, there was marked interlobular fibrosis, with extensive atrophy of the acini. There was diffuse lymphocytic infiltration of the fibrous tissue. The ducts were dilated and filled with small concretions. The duct of Wirsung was patent and opened into the duodenum by an aperture separate from that of the common bile duct. The duct of Santorini was not found. Osteoporosis of the ribs was present.

Case 2: J.F., male, age seven months, entered with complaint of chronic cough of five months duration. Two older brothers are well. Sister died of bronchopneumonia at six months of age, after having had chronic cough since the age of two months; she was always a feeding problem, had large foul-smelling stools, and necropsy showed multiple lung abscesses, pancreas not sectioned. Present patient was a feeding problem almost since birth, usually taking only two to three ounces at a feeding. There had been mucus in the stools since birth, and they were six to nine in number daily. He was breast-fed until six months, then was supplemented. It was noticed that the cod liver oil went through with the stools, undigested. The father, who was a physician, noticed that there was a large amount of undigested fat in the stools. The patient's cough, starting at the age of two months, remained moderate and unchanged for about three months, and then the child began wheezing. This gradually became worse, until during the two weeks before entry to the hospital he became dyspneic, using the accessory muscles of respiration, raising sputum, and became cyanotic on exertion or crying.

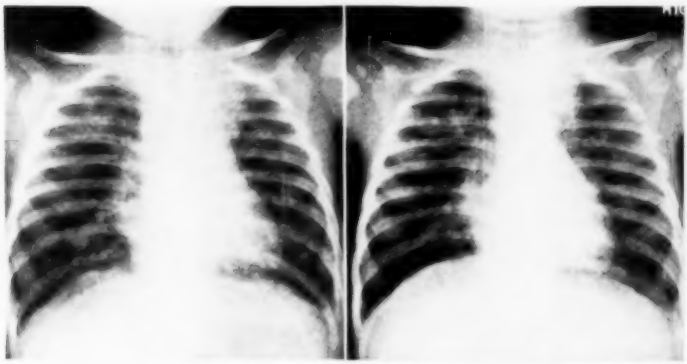

FIGURE 3 FIGURE 4

Fig. 3, Case 2: On admission, broncho-pneumonia and atelectasis in left upper lobes.—*Fig. 4, Case 2:* Seven days after Figure 3, and four days of treatment.

Sputum was mucopurulent in character. Physical signs appeared first in the chest at this time. Ephedrine gave no relief. No fever had been noted. Examination showed a baby breathing with stridor. Mucopus was present in the nasal passages and in the pharynx. The child was breathing with the accessory muscles of respiration. Coarse rales were present throughout the lung fields. Tuberculin and coccidioidin skin tests were negative. The white count varied between 15,000 and 18,000, with between 60 to 90 per cent polymorphonuclears. The hemoblobin was 68 per cent, with 3,790,000 red cells. Urine on entry showed one plus sugar. The stools showed large amounts of fat, and were bulky and foul smelling. Roentgen changes are seen in Figure 3, and in Figure 4 following treatment. Studies of sinuses showed the antra and ethmoids full. Blood level of Vitamin A was 3 I. U. per 100 cc. (control 28 I. U.); carotenoids were 0.01 mgm. per 100 cc. (control 0.18 mgm.). Sputum obtained by bronchoscopy showed heavy growth of Staphylococcus aureus (coagulase positive). Four days after admission 100,000 units of Vitamin A were given intramuscularly, and this was repeated in two days. Fifteen drops of percomorph oil were given daily, 5½ grams of pancreatin and two ounces of banana powder were added to a quart of milk to make the formula. The baby was given clysis, a transfusion and sulfathiazole, and improved rapidly clinically. Two days after first administration of intramuscular Vitamin A, the blood level of vitamin A rose to a low normal, but no carotenoids were present. In six days practically no fat was seen in the stools with Sudan III stain. The clinical improvement did not last, however, and the baby died 11 days after admission.

Necropsy report: The lungs weighed 200 grams together. The pleural surfaces were smooth. There were several large, angular, dark purplish sunken areas on the surfaces of both lungs. These corresponded to wedge-shaped, dark, red, firm, airless areas on the cut surface (Fig. 5). Multiple tiny, 1 mm., round, yellowish patches of exudate studded the cut surface of these areas. Occasional dilated thin-walled bronchi, filled with thick pus, were identified. Other portions of the lungs were emphysematous.

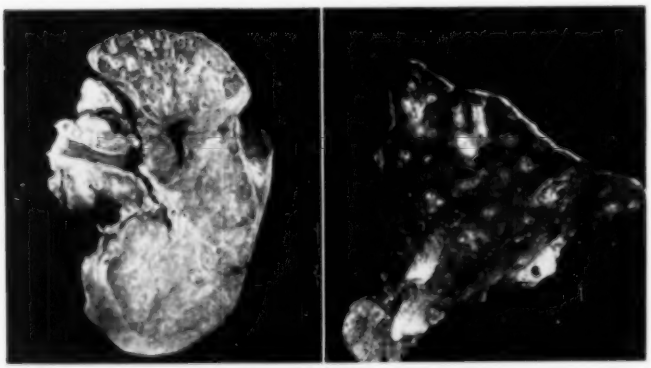

FIGURE 5a FIGURE 5b

Fig. 5, Case 2: Cut surfaces of left lung, chronic suppurative bronchitis, bronchiectasis in upper lobe, and marked enlargement of peribronchial nodes.

Histologically, there were large areas of atelectasis with perivascular and peribronchial fibrosis. Many dilated bronchi were filled with polymorphonuclear exudate and their walls were thickened, vascularized, and infiltrated with mononuclear cells. The epithelium of one of the bronchi of the left upper lobe was "heaped up," suggesting slight or early squamous metaplasia (Fig. 6). There were scattered patches of alveolar leucocytic exudate. The liver weighed 300 grams. Histologically, the parenchymal cells contained moderate fat deposits, especially in the central zones. The hepatic and common bile ducts were patent and bile-stained. The gall bladder contained clear, unstained mucoid material. The pancreas measured 6 x 2 x 1.5 cm. and weighed approximately 10 grams. The distal end reached only about half way from the loop of the duodenum to the hilus of the spleen. The pancreas cut with unusual resistance and the cut surface was composed of abnormal confluent lobules about 2 mm. each in diameter. Histologically, there was marked perilobular fibrosis with atrophy of the acini and moderate mononuclear infiltration. There were many dilated small ducts and ductules, many of which were filled with hyaline acidophilic concretions (Fig. 7). There was no metaplasia of the duct epithelium. The main pancreatic duct was patent and opened into the ampulla of Vater about 5 mm. proximal to the tip of the papilla. The accessory duct was not found. A small enterogenous cyst, 1 cm. in diameter, was found attached to the mesentery.

Case 3: B.M., female, age three months, entered with the complaint of difficult breathing and cough for two months, and failure to gain. One sibling died of bronchopneumonia at three months. The patient was born at eight months gestation, a breach presentation, weighing five pounds 10 ounces. The respiratory difficulty was first noticed when the child was first brought home from the hospital at the age of one month. Inspiratory and expiratory wheezing and retraction of the ribs was observed when the baby cried or was otherwise disturbed, and breathing seemed rapid. All of the symptoms became gradually more severe, and a dry cough, infrequent at first, increased to paroxysms occurring every 10 to 30 minutes during the day, and frequently wakened the patient at night. The last two weeks vomiting had occurred following the coughing. No fever or nasal discharge had been noted. Physical examination showed a small, fairly well nourished infant of about nine pounds in weight. There was continuous respiratory distress with marked retraction of the lower ribs. Coarse, inspiratory rales were present at the right base and over the whole of the left lung, more numerous in the axilla and back. The respiratory rate on entrance was seventy per minute. The heart was normal, but the rate was 180. The liver edge was palpable 3 cm. below the right costal margin. Blood Wassermann, and tuberculin and coccidioidin skin tests were negative. Initial blood counts and urine were normal. The sedimentation rate was 3 mm. per hour. The stools consistently showed large amounts of neutral fat, and were large, bulky and foul-smelling up to the time that the dietary regimen was instituted. Assay of duodenal juice for trypsin by Andersen and Early's modification of the Fermi method showed the equivalent of five viscosimetric units, or the minimal detectable tryptic activity. Throat culture showed a pure growth of hemolytic streptococci. Roentgenograms of the chest are shown in Figure 8.

Treatment consisted in a high-protein, high-caloric diet, low in fat, with much of the protein being given in the form of hydrolysed casein.

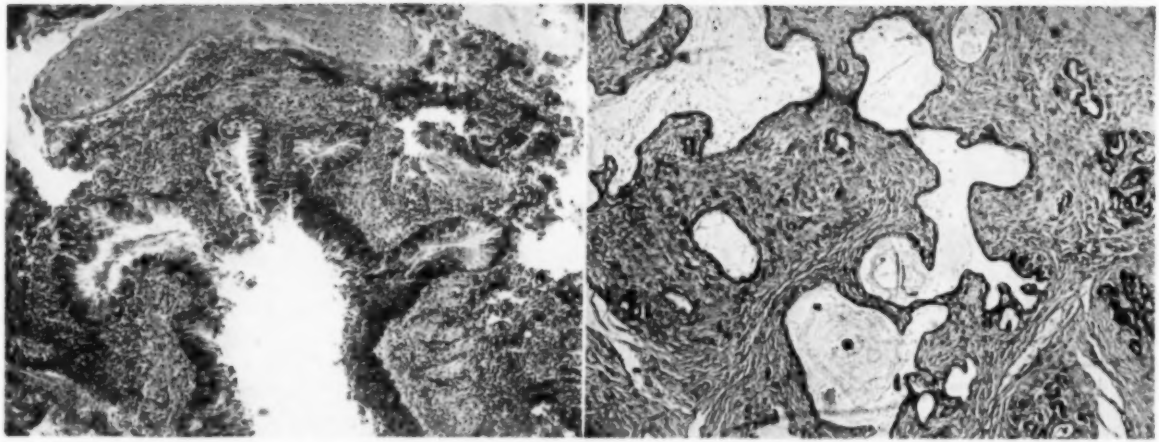

FIGURE 6

FIGURE 7

Fig. 6, Case 2: Section of bronchus from left lung showing metaplasia of epithelium.
Fig. 7, Case 2: Section of pancreas showing cystic dilatation of ducts.

A large part of the carbohydrate was given in the form of simple sugars; she received banana, both in the whole form, and as banana flakes added to the formula. The diet was supplemented with a multiple vitamin preparation carrying the fat-soluble vitamins in a water-soluble menstruum. Penicillin was started intramuscularly, and later given also as an aerosol inhalation (50,000 units every three hours). This child was kept in the hospital for 14 months, the course being slowly, but steadily favorable. The fat content of the stools dropped off sharply, and finally the stool contained none at all. Gain of weight was steady. Moderate dyspnea continued and rales could be heard for six months. One attempt at discontinuing the penicillin resulted in severe respiratory distress, so penicillin aerosol was resumed. Streptomycin aerosol was added during the latter months of her stay, as the Staphylococcus aureus in her sputum was replaced by a penicillin-resistant E. coli. She was dismissed in good condition on the prescribed diet, 50,000 units of penicillin aerosol twice daily for five days a week, and 50 mgm. of streptomycin aerosol twice daily for two days a week. Roentgenograms during her stay in the hospital are shown in Figures 9 and 10. She is well a year after dismissal, but still taking treatments.

Case 4: L.G., male, age 14 years, entered with the complaint of chronic cough of 10 years duration. The family history was entirely negative. Birth was normal, a breach presentation being corrected before delivery. Birth weight was nine pounds. Patient was well until the age of 10 months, when he began having large, loose and foul-smelling stools, three to seven daily, in number. These continued until the age of nine years when he had an attack of acute abdominal pain, and an appendectomy. Since the age of four there were frequent head and chest colds. Acute sinusitis occurred at four and five years, and pneumonia was diagnosed three times. Nasal polyps were removed at the age of four. Allergy to many grasses and a few trees were proved and improvement was noted for a time under desensitization. Clubbing of the fingers has been noticed for at least the last three years. The appetite has always been excellent, but he has always been very much underweight. He had no fever except during acute respiratory episodes. Roentgenograms taken during allergy investigation three years previous to entry are shown in Figure 11. At this time the antra, ethmoids and frontals were completely filled with material of soft tissue or fluid density, and the sputum showed a heavy growth of Staphylococcus aureus, coagulase positive. No fat was present in the stools at this time. Physical examination at time of entry showed a thin and underweight boy, but active and alert. Polyps were present in the nasal passage. Respiratory rate was 30 per minute. There were fine scattered rales in the bases of both lungs. There was distinct clubbing of the fingers, and the nail-beds were moderately cyanotic. The skin was dry. Blood Wassermann, and tuberculin and coccidioidin skin tests were negative. White blood count was 18,000, with 70 per cent polymorphonuclears. The red cells were normal, sedimentation rate was 10 mm. per hour. Urine was normal. The stools were large, foul, with an enormous amount of fat. Duodenal juice showed only the smallest trace of trypsin. Sputum showed large numbers of Staphylococcus aureus, coagulase positive. Roentgenograms of the sinuses showed bilateral clouding of the antra, anterior ethmoids, and of the frontals, and those of the chest are shown in Figure 12. Bronchograms did not reveal any positive evidence of bronchiectasis (Fig. 13).

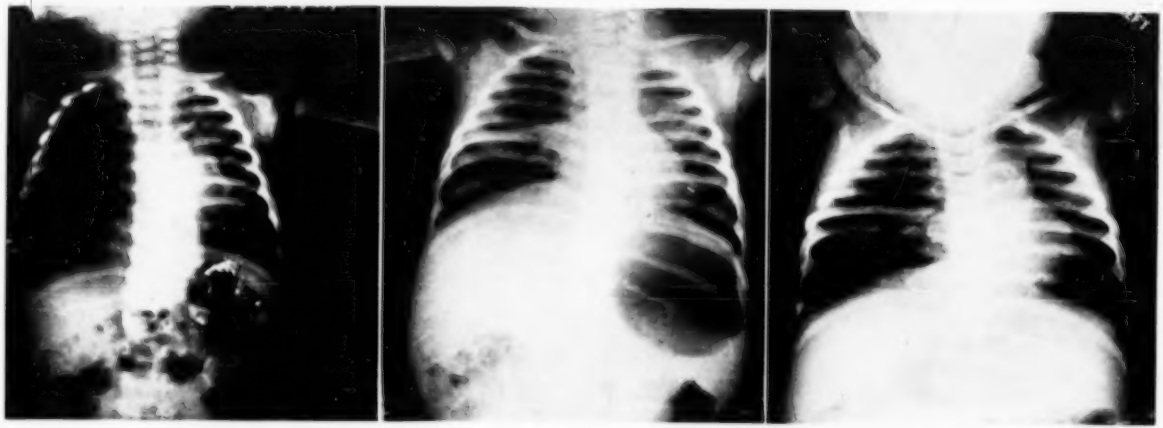

FIGURE 10 FIGURE 9 FIGURE 8

Fig. 8, Case 3: Chest on admission, broncho-pneumonia and atelectasis on left, with shifting of heart to that side.

Fig. 9, Case 3: Ten weeks after admission, atelectasis now present on right side.

Fig. 10, Case 3: Six months after admission, rales have disappeared from chest.

Treatment consisted first of surgical removal of the nasal polyps. He was placed on a high-caloric, high-protein, low-fat diet, with a multiple vitamin preparation with the fat soluble vitamins in a water-soluble menstruum. Aerosol penicillin was given in daily doses of 300,000 units, with prompt clearing of chest, and abatement of respiratory symptoms and signs. Because with the disappearance of the Staphylococcus aureus in the sputum there appeared a penicillin-resistant Pseudomonas aeruginosa, streptomycin was also given by aerosol, and he was dismissed on this treatment. At the age of 14½ years he is clinically in excellent condition, with no symptoms or signs referable to the chest.

The four case histories given above are representative of the series of 10 cases seen on our Pediatric service in the last seven years. There were six male, and four female patients. Four had had symptoms from birth, two since the age of one month, one since two months, one since 10 months, and two since the age of 14 months. In five the initial symptom that brought the patient to the physician was a cough, in five it was the character of the stool. The latter group all developed cough and respiratory symptoms later, with one exception having nasal discharge and otitis media within a year. Of the four who developed cough following the typical stools, it followed at intervals of 14 months, three, four, and five years. In three cases there was a history of a sibling dying previously with a clinically similar disease, and in one of a sibling with the typical stools in infancy who recovered spontaneously. In seven of the 10 cases typical stools were present, in three there was at least a distinct increase of fat in the stools, in two of which there were no other stool abnormalities, and in one blood was noted. In nine there was a polymorphonuclear leukocytosis of 15,000—30,000 on entry to the hospital, in one there was a count of 10,000. In the two cases that died and were proved by necropsy, no trypsin determinations were done; four others showed no trypsin activity in the duodenal contents, and four showed minimal activity only. In all 10 patients the tuberculin and coccidioidin skin tests were negative. There was marked evidence of paranasal sinusitis by examination and roentgenograms in four, one of whom had recurrent polyposis, and one mastoiditis. Three had marked and heavy chronic nasal discharge, two others postnasal pus, and one had no evidence of sinusitis. There were three who had otitis media, including the one listed as having no evidence of sinusitis. From the sputum of seven were obtained cultures of Staphylococcus aureus, coagulase positive, one cultured hemolytic streptococci, and in two no pathogenic organisms were found in bronchial secretions. In the patient having hemolytic streptococci, E. coli was found after the former had disappeared, and in one in whom the Staphylococcus aureus disappeared after treatment, Pseudmonas aeruginosa appeared later. On physical

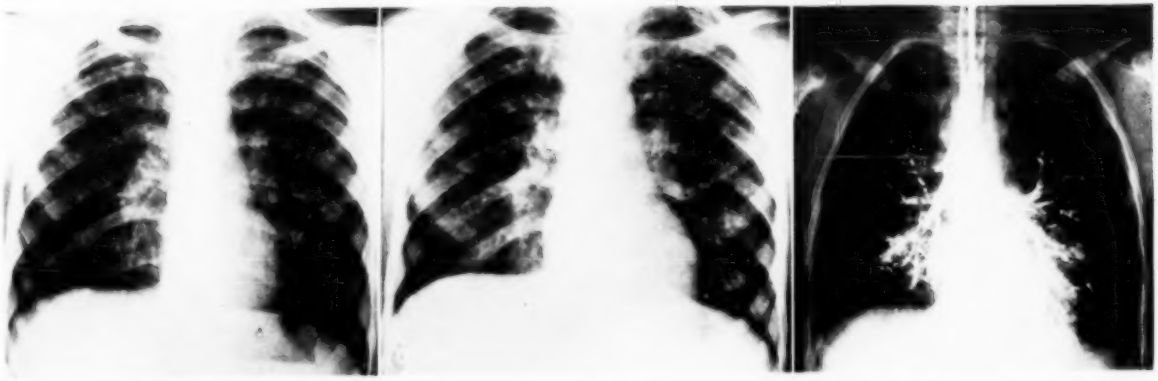

FIGURE 11 FIGURE 12 FIGURE 13

Fig. 11, Case 4: Chest film three years before hospital admission. Scattered densities and heavy hilar markings.

Fig. 12, Case 4: Chest film on admission, three years after Figure 11. Increased densities.

Fig. 13, Case 4: Chest film, lower lobes free of bronchiectasis.

examination all showed malnutrition except one, all had physical findings in the chest, and all but one marked involvement by roentgen studies.

The first two patients in this series, both seen before the use of antibiotics was available, died at the age of seven and 27 months. In both, in addition to fibrocystic disease of the pancreas, there was demonstrated extensive pulmonary disease consisting of bronchitis, pulmonary abscesses, bronchiectasis, and bronchopneumonia. With the introduction and availability of the antibiotic drugs, all but one of the succeeding eight cases have been saved. Three patients are well, three are in fairly good health, and one, though not in good health is being kept quite comfortable by the use of penicillin and streptomycin, and is not in a serious condition. The one who died left our clinic and treatment was not continued. Of the seven survivors there is one each of two, two and a half, three, and five and a half years, two of seven years, and one of 14½ years. All 10 of our patients were members of the white race.

Discussion

Our knowledge of cystic fibrosis of the pancreas and its associated lesions as an entity is fairly recent, and an excellent history of the disease may be found in the account of Andersen.[1] It is much more common than formerly thought to be, and was found in about 3 per cent of the necropsies at the Babies Hospital in New York. In 60 per cent of these the respiratory symptoms and pulmonary disease had masked the pancreatic disease. Farber[2] reported 4.8 per cent between 1937 and 1942, but an incidence of 12 per cent in 1942.

The typical symptoms have all been mentioned in the case histories presented. They are chiefly those of early failure to gain on an adequate diet, with unimpaired appetite, with steatorrhea, soon followed by symptoms of chronic respiratory infection. The latter are the symptoms for which the patient is often brought first to the physician, and they very soon dominate the clinical picture. The cough resembles that of pertussis and this disease is often suspected. The thick tenacious mucus is similar in consistency to that found in whooping cough. Sometimes conjunctivitis is present, although it was not observed in our series. The child is underweight and there may be abdominal distension.

It occurs quite often in other members of the family. On physical examination signs of infection are found usually along the entire respiratory tract, practically always in the lungs. Staphylococcus aureus is usually found in cultures of the respiratory secretions, other pathogenic organisms occasionally. Those originally found may be replaced by penicillin-resistant organisms after the use

of this drug. A distinct polymorphonuclear leukocytosis is usually present before the beginning of treatment. An examination of the duodenal contents always shows absent or greatly diminished trypsin activity, and is always necessary to make certain the diagnosis. This examination should be repeated at several intervals for substantiation. The test for trypsin activity by placing an emulsion of stool on x-ray film is of some value when trypsin analysis of the duodenal contents is not technically available, but it should not replace the more accurate test. The vitamin A absorption curves are flat in this disease.

The roentgenological findings are extensive. Barium in the intestinal tract may reveal complete obstruction during the newly-born period if the mucus in the meconium is viscid enough to cause the condition known as meconium ileus. This condition is fatal and is present before pulmonary disease is manifest. If the condition be less severe, the typical picture of vitamin deficiency may show in the intestinal tract as "laking," with air and fluid levels and markedly slow bowel motility. Loops are dilated, and the distal small bowel is obviously smaller than the distended portions. Neuhauser[3] describes the extensive pulmonary changes and divides them into two stages. In the first there is evidence of bronchial plugging without much infection. There is flattening of the diaphragm, and many portions of the lung will appear emphysematous. There may be bulging of a few or many of the intercostal spaces. The lungs remain hyperexpanded in expiration, and the picture of obstructive emphysema with poor air exchange may be recognized. Areas of lobular atelectasis may be present, and lobar collapse is frequent. The second stage shows infection supervening, with increased hilar shadows and prominence of the bronchovascular markings. Thickened bronchial walls may occasionally be seen and areas of peribronchial increased density. Although bronchiectasis is usually present, Neuhauser does not believe lipiodol is warranted to confirm this. The patient with long-standing pulmonary disease shows irregularly emphysematous lungs with prominent hilar shadows and bronchovascular markings, extensive peribronchial pneumonia, areas of atelectasis, bronchiectasis, and bronchiectatic abscesses. The apices and bases are equally involved, and the infiltration often extends to the periphery of the lung with no evidence of pleural reaction. All of these changes are not specific, but should suggest the diagnosis.

Among the diseases to be included in differential diagnosis, coeliac disease resembles pancreatic fibrosis the closest, and is more apt to be confused than others. Although the character of the stools is quite similar, coeliac disease appears later in infancy, pulmonary symptoms are usually not outstanding, and trypsin is

not absent or markedly diminished in amount in the duodenal contents. Allergic asthma, because of the wheezing, and lipoid and unresolved pneumococcic pneumonias, may be confused. These, however, may be differentiated by careful history, and again by duodenal content analysis. The chronic specific infections such as tuberculosis, coccidiomycosis, pertussis and syphilis may be differentiated by the specific tests for these diseases, as well as by analysis of the duodenal contents, and any one of these diseases may occur during the course of pancreatic fibrosis. As always, enlarged thymus and foreign body in the respiratory tract may be suggested, at least by the parents, as possibilities.

The pathological conditions found in cases coming to necropsy are extensive. There is always a fibrotic or cystic pancreas in which the ducts may be open or in a state of atresia. Either small multiple abscesses, bronchiectasis often more marked in the upper lobes, or chronic pneumonia, or combinations of these are present. Osteoporosis may be present if the patient lives into the second year, perhaps dependent upon deficient calcium and vitamin D absorption. This occurred in one of our cases (Case 1). True rickets is usually absent probably because of the slowness of growth.

There are several interesting theories as to the cause of this disease. Andersen and Hodges,[4] because of the hereditary trait which they believe to be present, conclude that cystic fibrosis of the pancreas is part of a hereditary disease also occasionally involving the intestinal glands, intrahepatic bile ducts, gallbladder and other epithelial glands. The primary feature is the production of abnormal secretion, with the morphological changes secondary. They believe the pulmonary disease primarily due to dietary factors, partially but not purely to lack of vitamin A. Baggenstoss, Power, and Grindlay,[5] have suggested a congenital deficiency of secretion as the primary cause of a diminution of pancreatic function. Both they and Andersen and Hodges believe the pulmonary disease to be secondary to the nutritional disturbances, including vitamin A deficiency which would result from pancreatic exocrine deficiency. Against the theory of the pulmonary lesions being dependent on a vitamin A deficiency is the fact that the pulmonary lesions do not disappear with the giving of vitamin A intramuscularly, and that the pulmonary disease appears too early for this to be plausible unless it presupposes a vitamin A deficiency of the mother. Lesions due to vitamin A deficiency are not found extensively at necropsy. Obstruction by an abnormal secretion of mucous glands is the theory accepted by many and upheld chiefly by Farber.[6] He has demonstrated that the secretion obtained by duodenal drainage is thick and inspissated and has stated that dilatation of glands and inspissation of secretion in

the trachea, bronchi, esophagus, duodenum, gallbladder, salivary glands and jejunum can be shown. Other surrent and less plausible theories are discussed by Wiglesworth.[7]

The treatment of this disease should be early and vigorous. The diet should be dictated by the pancreatic deficiency, and lies within the province of the pediatrician or the general practitioner. Large amounts of vitamin A should be given by mouth in a water soluble form, and intramuscular therapy with vitamins is not necessary except possibly initially. If pulmonary symptoms are marked when the patient is first seen, the situation may be a matter of immediate survival. It is often necessary to administer oxygen, and to remove the tenacious bronchial secretions by mechanical suction. Both the immediate and long-time treatment of the pulmonary complications should consist of the giving of penicillin. Aerosol is the method of choice, although this may offer some difficulties in very small infants due to inability to cooperate. The intramuscular route should be used if aerosol is not feasable, and may be used in addition at any time. It should be given in adequate doses until such time as the respiratory tract is clear of disease, and to some extent prophylactically during remissions. Streptomycin is valuable when penicillin-resistant organisms have replaced the original invaders. We have not had to use aureomycin or the other newer antibiotics, but they should be tried when penicillin and streptomycin are found inadequate. It is possible that aureomycin may ultimately prove to be the drug of choice in this disease, although its administration may offer technical difficulties in small infants. Local treatment, sometimes surgical, is occasionally needed for the disease in the sinuses. Full treatment should be reestablished immediately upon any return of symptoms. Avoidance of exposure to infection should be rigid at all times, and all prophylactic measures used against such diseases as measles and pertussis.

It is possible that an occasional spontaneous recovery may occur, but they have not been recorded. An occasional case may go unrecognized for some time if the disease be mild. However, the pathological changes in the respiratory system may in part be irreversible. Obviously the most important single item in the treatment of the disease is its early recognition, the burden of this lying with the pediatrician and the general practitioner, and occasionally with the chest specialist who may be called into consultation.

CONCLUSIONS

1) A series of 10 cases of the pulmonary complications associated with cystic fibrosis of the pancreas is reported.

2) It is a fairly common disease, and probably accounts for at least 4 per cent of all deaths in children.

3) A history of similar disease in siblings is fairly common.

4) The respiratory signs and symptoms soon dominate the clinical picture in this entity, and it should be suspected in any infant or young patient with steatorrhea and a chronic cough.

5) The differential diagnosis is made complete by the examination of the duodenal contents and the finding of an absent or minimal trypsin activity on repeated examinations.

6) Early recognition is important to prevent irreversible changes in the pulmonary tissue, and prognosis is improving with increased knowledge of the disease. In the present series of 10 cases seven are alive, and the treatment has been successful in six patients, only one patient having died since the use of antibiotics.

7) Paranasal sinusitis is usually an associated condition.

8) Because Stpahylococcus aureus is usually present in the respiratory secretions, penicillin by aerosol is the drug of choice, and streptomycin should be added if other pathogenic organisms which are penicillin-resistant replace the original invaders. Aureomycin and other antibiotics will probably soon have a place in the treatment of this disease.

9) Vigorous prophylaxis should be practiced against the common cold and its complications, and against other specific respiratory infections, especially measles and pertussis. The patients should be kept under constant supervision at all times.

CONCLUSIONES

1) Se informa sobre una serie de 10 casos de las complicaciones pulmonares que acompañan a la fibrosis quística del páncreas.

2) La enfermedad es bastante común y probablemente causa el 4 por ciento, por lo menos, de todas las muertes en la niñez.

3) Frecuentemente se obtiene la historia de una enfermedad semejante en los hermanos de padre y madre.

4) Los signos y síntomas respiratorios predominan pronto en el cuadro clínico de esta enfermedad, que debe ser sospechada en todo niñito o enfermo joven con esteatorrea y tos crónica.

5) Se completa el diagnóstico diferencial con el análisis del contenido duodenal y el hallazgo de que la acción de la tripsina está ausente por completo o es mínima.

6) Es importante reconocer este estado oportunamente a fin de evitar alteraciones irreversibles en el tejido pulmonar. Está resultando más halagüeño el pronóstico con el mejor conocimiento de la enfermedad. En la presente serie de 10 casos, siete están vivos. Ha obtenido buen éxito el tratamiento en seis pacientes, sólo uno ha muerto desde que se comenzaron a usar los antibióticos.

7) La senositis paranasal generalmente acompaña a este estado.

8) Debido a que se encuentra generalmente el estafilococo áureo en las secreciones respiratorias, la penicilina por aerosol es la droga de elección, y debe añadirse la estreptomicina si microbios patógenos resistentes a la penicilina reemplazan a los invasores iniciales. Es probable que la áureomicina y otros antibióticos pronto obtendrán un lugar en el tratamiento de esta enfermedad.

9) Debe emplearse una vigorosa profilaxia contra los resfriados y sus complicaciones, lo mismo que contra otras infecciones respiratorias específicas, especialmente la alfombrilla y la tos ferina. Deben mantenerse a los pacientes bajo una supervigilancia constante.

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Discussion

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Cystic fibrosis of the pancreas is a condition well known to all pediatricians. To those of us who have had the opportunity of observing and following children suffering from it, the results presented by Dr. Dickey are especially gratifying.

Until the use of antibiotics the general experience had been that the prognosis was a poor one, death usually resulting from purulent bronchitis, bronchopneumonia or bronchiectasis.

As Dr. Dickey clearly stated, this disease presents a symptom complex of a lack of pancreatic secretion, a low level of vitamin A in the serum, and pulmonary susceptibility to infections resulting in most cases in bronchiectasis.

Just what factor or factors are there responsible for this symptom complex? What connection is there between a condition of

bronchiectasis and a fibrosis of the pancreas which prevents the proper flow or formation of pancreatic secretion?

As Dr. Dickey mentioned, we know that the lack of pancreatic secretion prevents the digestion of fats and accordingly prevents the absorption of vitamin A, a fat soluble vitamin.

It has been shown by Bodansky and his co-workers that in cystic fibrosis of the pancreas, as well as in patients with a complete obstruction of the bile duct, the absorption curve from oily preparations of vitamin A is flat. However, when aqueous preparations of vitamin A were given the vitamin A curve rises to over 5000 units per 100 cc. plasma.

But in this lack of adequate vitamin A the factor responsible for the bronchiectasis? This does not appear to be so.

Experimentally the lack of vitamin A brings on Xerophthalmia and night blindness but does not cause bronchiectasis. In certain countries such as China, India, the Dutch East Indies and parts of Africa there is general lack of adequate vitamin A among the people—but as far as can be determined they have not an unusually high incidence of bronchiectasis.

Andersen, however, while not claiming the lack of vitamin A as the causative factor in the production of bronchiectasis nevertheless claims that the nutritional difficulties and the infections of the respiratory tract are closely related. She has stated that in a few patients who received dietary therapy prior to the onset of the chronic cough, respiratory infections did not occur. When the respiratory infections were well established, dietary therapy was rarely effective.

In 1948 at the Annual Convention of the American Academy of Pediatrics, Dr. Andersen presented a series of 107 cases of cystic fibrosis of the pancreas which she had followed—of these 72 were still alive as of that date. Of 79 who received penicillin therapy 62 were living of which 22 had no evidence of coughing. It is to be noted that Dr. Andersen gave penicillin parenterally as well as by aerosol in cases that showed evidence of acute pneumonitis.

There is evidence that heredity is of etiological importance but this too is not the general rule.

There must be some other factor responsible for this symptom complex of which we have no knowledge at the present time.

It would seem that this factor is capable of producing an increased viscosity of glandular secretions in the respiratory as well as in the gastro-intestinal tract.

This extremely thick mucus causes an obstruction to the bronchi and bronchioles with the subsequent formation of bronchiectasis.

Farber suggested calling this whole symptom complex mucoviscidosis. It has been suggested that some factor similar to an

Rh substance may be injected and inhaled by infants from the ammotic fluid before birth and that the developing glands in the respiratory and intestinal tracts have undergone damage. This also has not been demonstrated.

Dr. Dickey has pointed out the method for diagnosis of cystic fibrosis of the pancreas—the examination of duodenal contents for estimation of amount of trypsin. He has described the appearance of these children and their digestive symptoms.

He has demonstrated that a disease, considered as fatal only a few years ago, may be relieved in the majority of cases by proper dietary care, by aerosol therapy and by avoiding as much as possible contacts with colds and other respiratory infections.

HARRY SHWACHMAN, M.D.
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Dr. Dickey and Dr. Levine have presented a clear and thorough treatment of a disease which has been recognized with increasing frequency not only in pediatric institutions but also in chest clinics in this country and many other countries. Our experiences in Boston are similar in many respects to those of Dr. Dickey in San Francisco.

A reference was made to the development of a simple screening test employing unexposed x-ray film for the detection of tryptic activity in the stools (Shwachman, Patterson, Laguna, *Pediatrics*, In press). The test has been found useful because of its simplicity and above all by the fact that it can be performed in the physician's office. A stool emulsion 1:5, 1:10 in water or sodium bicarbonate is made and a drop is placed on the film and incubated at 37 degrees C for one hour. If trypsin is present the gelatin will be digested and a clear space will appear. Patients with this disease usually have no trypsin in their stools on repeated examinations and when given oral pancreatin the test becomes positive, if the dose is sufficient. To illustrate the viscid nature of the material secreted by these patients, a photograph of the viscid duodenal fluid is shown. Not only is trypsin absent but the other pancreatic enzymes, lipase and amylase, are absent as well. The administration of secretin to a patient with this disease will not increase the flow of pancreatic juice nor cause an out-pouring of enzymes. I should like to register my objection to the terms cystic fibrosis of the pancreas, and pancreatic fibrosis, because we are not dealing with a disease of the pancreas alone but rather with a generalized disease which may involve lungs, liver and many other organs in varying degrees. Reference has already been made by Dr. Farber's term "muco-viscidosis" to signify the essential alteration in mucous secreting glands as a primary defect.

This disease must be differentiated from those conditions in which pancreatic duct obstruction is produced by congenital anomalies or by masses or stones as seen commonly in the adult patient.

Dr. Dickey referred to a number of conditions which have been confused with pancreatic fibrosis or "muco-viscidosis." I should like to present a list of diagnoses made at the time of admission in some of our patients who later were shown to have pancreatic fibrosis.

NEWBORN:

Congenital Anomalies,

atresia of stenosis or any condition producing obstruction in this age period.

INFANTS AND YOUNG CHILDREN:

Pulmonary,

chronic respiratory infection,
pertussis,
asthma,
empyema (usually staphylococcal),
bronchopneumonia, chronic,
bronchiectasis,
atalectasis,
lipoid pneumonia,
tuberculosis.

Nutritional and Gastrointestinal,

marasmus,
food intolerance,
celiac syndrome,
a) congenital anomalies of
1. intestine or mesentery,
2. other organ systems.
b) infection,
1. parenteral—lungs in particular,
2. enteral—tuberculosis, syphilis, dysentery, parasites,
3. celiac syndrome idiopathic.

OLDER CHILDREN:

Bronchiectasis.

In one respect our recent experience differs from that of Dr. Dickey and the writings of the literature. We rarely have difficulty in differentiating idiopathic celiac disease from pancreatic fibrosis —two conditions that differ so much that one wonders how the original confusion arose so often.

That we are improving our diagnostic acumen can be seen from the following table:

DIAGNOSIS NOT MADE PRIOR TO POST-MORTEM

Prior to 1938	100 per cent
1938 - 1942	41 per cent
1943 - 1947	25 per cent

Post-Mortem Incidence

1933 - 1947	3.6 per cent of 2,959 autopsies

This table includes Dr. Farber's post-mortem figures on the incidence of this disease in routine examinations. The incidence of this disease in Boston is high as seen from the following data:

CLINICAL INCIDENCE

1938 Condition first diagnosed on Infants Ward and confirmed at post-mortem.

1947 Roster of living patients approximately 40.

1949 Roster of living patients approximately 85.
51 new cases; diagnosis confirmed by duodenal intubation in past 18 months.

Although the majority of patients are brought to the physician during early infancy because of pulmonary involvement and failure to gain in weight, relatively few of the older children are referred with bronchiectasis. Our oldest patient, the chest film of which is shown on the slide, died at the age of 12 with a six year history of bronchiectasis. The diagnosis of pancreatic insufficiency was made approximately one year before death although this disease might have been suspected because of abnormal stools and failure to gain in early infancy.

Dr. Dickey referred to the frequency with which the *staphylococcus aureus* is obtained from the nasopharynx of these patients. I should like to emphasize this observation and mention a nearly 100 per cent recovery of this organism from the nasopharynx of the last 150 patients studied by Mr. Foley and myself.

The remarkable therapeutic results achieved by the use of antibiotics as pointed out by Dr. Dickey points to the importance of the pulmonary infection and its early recognition. The use of aerosol penicillin and streptomycin as well as by the intramuscular route, along with dietary and pancreatin replacement therapy, has made it possible to save many of these patients. That aureomycin given in one daily oral dose is effective and practical is the subject of a report which will appear in the New England Journal of Medicine (Schwachman, Crocker, Foley and Patterson, In press). The following summary is presented:

RESULTS OF AUREOMYCIN THERAPY

Cases	50 (Based on observations from two months to six months)	
Age range	5 weeks to 12 years	
	50 per cent of cases over 2 years of age	
Onset of pulmonary symptoms,		
	under 3 months	27 cases
	between 3 months and 2 years	15 cases
	over 2 years	8 cases
Condition when aureomycin started,		
	very poor	9 cases
	poor	21 cases
	fair	13 cases
	good	7 cases
Dose	20 to 30 mg. kilo. day, given in one or two doses	
Results	excellent to good	45 cases
	unsuccessful	5 cases

I should like to show my last slide of an infant with marked malnutrition at the ages of five and seven months, and again at two and one half years. This remarkable improvement has been achieved by the use of the combined dietary, antibiotic, and replacement pancreatin therapy.

Whether these children will now be able to survive and reach adulthood or whether some of your young patients with bronchiectasis have this disease remains to be seen. I sincerely hope that with the aid of men in your society great advances in the understanding of the nature of this disease may be made.

The Surgical Management of Lung Abscess*

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Any present day discussion of the surgical treatment of lung abscesses must first evaluate the impact of chemotherapy and antibiotics on the lung abscess problem. These drugs not only have changed the surgical concepts but have altered the type of abscess encountered. The incidence of abscess and empyema has decreased with the effective treatment of predisposing pneumonias, but those abscesses which do develop still present serious problems.

Present day abscesses are more likely to be chronic and complicated, allowed to coast past the optimal time for drainage because medical treatment can control the toxic symptoms. With the use of antibiotics emergency surgical drainage is seldom needed and definitive surgery usually can be withheld until the patient is a better candidate.

The earlier sulfone drugs were of value in the pneumonias, undoubtedly preventing many abscesses but curing few. Penicillin will cure acute aerobic abscesses, but all forms of chemotherapy are ineffective in chronic abscesses. By controlling toxic symptoms and eliminating the foul odor the antibiotics actually foster procrastination of necessary surgery, occasionally disguise a carcinoma of the lung, and allow chronic abscesses to develop. An abscess which has been kept relatively sterile for months is not cured; the structural disease persists and signs of toxicity will resume as soon as the drug is stopped. If irreversible fibrotic or bronchiectatic changes have occurred or if the abscess has become multiloculated simple drainage is then ineffective and resection of lung tissue is necessary.

The present trend is toward overtreating with antibiotics and then resecting the abscessed lobe when it is obvious that conservative measures have failed. The need for surgical drainage is being narrowed,—not so much from the choice of the surgeon as by the stage of the abscess when it is presented for surgical consultation. It is the purpose of this paper to attempt to evaluate

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this trend by presenting the 247 cases of lung abscess we have treated during the past six years.

The published statistics on lung abscess have often been too inclusive to show the effects of any treatment, being based on the total number of cases diagnosed at a hospital within a given period. Such series include cases discovered at autopsy, moribund cases admitted in extremis, and abscesses secondary to carcinoma. A selected group, including only cases which have had a minimal test of treatment, presents a more accurate insight into the therapeutic problems,—even though this may be at the expense of distortion of the complete picture of lung abscess as a medical disease.

This paper is based on such a series of 247 lung abscesses treated within the six year period ending March 1, 1949. All cases were referred for surgical management, all received chemotherapy in some form, and every case had at least one bronchoscopy. Excluded were abscesses which proved to be secondary to carcinoma, bronchiectasis which was not due to an abscess, septic infarcts with abscess formation, and moribund cases which died shortly after admission and before effectual therapy could be established. This deliberate selection allows consideration of the curative value of the various forms of management yet gives the entire group the same base of chemotherapy and bronchoscopy.

Of the 247 cases 188 can be classed as cured, with from six months to six years follow-up by x-ray and clinical examinations in most cases. Thirty-four patients died, a mortality rate of 13.7 per cent for the entire group (Table 1). Every patient received chemotherapy in some form. The earlier cases had only sulfones; the more recent ones have had sulfones and antibiotics, getting

TABLE 1

Results in 247 Lung Abscesses — 1943-1949
(All had Bronchoscopy + Chemotherapy)

A. Chemotherapy + Bronchoscopy only			70
Cured 58 (82.8 per cent)	Dead 5 (7 per cent)		
B. Surgical Drainage			119
Cured 80 (67.2 per cent	Persisting disease	5	
Dead 25 (21 per cent)	Later resection	9	
C. Pulmonary Resection			58
Cured 50 (86.2 per cent)	Lobectomy	41	
Dead 4 (6.9 per cent)	Pneumonectomy	17	
Empyema present 4			
TOTAL			247

penicillin and/or streptomycin by parenteral and aerosol methods. Few cases are referred for surgical treatment who have not already had some antibiotics; surgical advice is usually sought only after it has long been apparent that chemotherapy is ineffective.

Seventy cases received only chemotherapy and bronchoscopy. Fifty-eight of these became well and five (7.1 per cent) died. Seven were not cured, refused drainage or lobectomy, and still have disease.

Surgical drainage was done in 119 patients. Single stage drainage was possible in 79, the remaining 40 had two or more stages. Of the drained cases 80 (67.2 per cent) were apparently cured by the procedure, although 17 required subsequent plastic closure of persisting bronchial fistulae. Twenty-five patients died (21.0 per cent). Fourteen were improved by drainage but not cured, nine of these having lobectomies because of residual abscess or bronchiectasis. Five have persisting disease which will require resection in the future. In order to tabulate the nine cases which were drained and later had lobectomy it seemed desirable that each of these patients be treated as two separate procedures, classified first as a failure of drainage, then as a cure by lobectomy. By this arbitrary assignment the 247 abscesses being reported are actually confined to 238 patients, nine of them having been subjected to two separate types of surgery a year or more apart.

Pulmonary resection was done in 58 cases. Forty had lobectomy and 17 pneumonectomy. Four patients died (6.8 per cent); three of these were in pneumonectomy patients. Fifty patients were entirely cured (89 per cent) and four were cured of the abscess but still have persisting empyema which has not yet healed.

An analysis of the causes of death is given in Table 2. The mortality of resection or drainage is far less than would be ex-

TABLE 2

Causes of Death in 34 Cases of Lung Abscess

Non-Surgical Cases		5	Resected Cases		4
Toxicity	3		Post Op. Shock	3	(2-P 1-L)
Hemorrhage	1		Empyema	1	(P)
Brain Abscess	1				
Surgical Drainage		25			
Toxicity	9		Empyema	2	
Hemorrhage	6		Esophageal Fistula	1	
Pneumonia	3		Disease of Opposite Lung	1	
Brain Abscess	3				

pected by medical management alone because all of the operative cases had already failed to respond satisfactorily to preliminary conservative management.

Standardizing the management of abscesses is difficult because the surgeon seldom sees and follows the disease from its beginning. Most cases referred for surgery already have chronic abscesses. In discussing the surgical treatment, therefore, some recognition must be taken of the phase in which the abscess is seen, whether it is 1) very early, in the "formative" state which is usually not referred for surgery, 2) an acute simple unilocular abscess, or 3) if it is a complicated or multilocular abscess.

1) In the acute "formative" abscess, a localizing pneumonitis with little cavitation or a circumscribed necrotic area, conservative methods should be used. Usually chemotherapy will have been started long before the roentgenologic diagnosis is apparent. These cases are given penicillin by parenteral and aerosol methods. Sulfadiazine is given concomitantly. Streptomycin was used in a few of our cases but did not appear to be more effective. Bronchoscopic examination is done in all cases, both for possible therapeutic effects and to eliminate the chance of neoplasm or foreign bodies as etiologic agents. Supportive treatment consists of bed rest, transfusions as indicated, vitamins, and iron. Postural drainage is occasionally effective in helping to keep the lower respiratory tract free of pus, but in most cases it seems to have little benefit and need not be persisted if no effectual drainage results.

2) In the acute simple abscess which is unilocular, especially if preliminary attempts at chemotherapy have shown no clinical or roentgenologic benefits, the treatment of choice is early drainage. Chemotherapy and bronchoscopy should precede drainage, but no abscess ought to remain undrained if clinical and roentgenologic signs show the disease to be progressing. Weekly roentgen examinations are made during the "conservative" management, and if a plateau occurs in the progress drainage is instituted. The surgical drainage is done in one stage if the pleura is adherent and the abscess has been correctly localized. If the pleura is not firmly adherent and indurated two stages are used. In our series of 119 drained cases 79 (64.7 per cent) were done as single operations. The advantages of a single stage are apparent, yet there are times when it is unsafe to drain an abscess across a poorly adherent pleura. If the abscess faces an interlobar fissure, or if it lies along the mediastinum, the peripheral zone of the affected lung segment may be adherent to other structures but not to the chest wall. Such cases cannot be drained as a single stage operation.

3) In complicated chronic or multilocular abscesses, where there is extensive lung damage, fibrosis, and bronchiectasis, resection of the involved lung tissue is preferable. Drainage of these cases is usually inadequate, unsatisfactory, and does not cure. The morbidity and mortality of pulmonary resection is much less than that of inadequately drained abscesses. In putrid abscesses with severe toxicity and profuse sputum it is sometimes safer to drain the abscess first, following with lobectomy after the toxic course has been checked and a comparatively dry bronchial tree is present at the time of resection. Such emergency drainages are infrequent since the use of penicillin.

Lung resection has become safe because of surgical, anesthetic, and blood replacement developments and the discovery of antibiotics to prevent and control pleural infection. These advances have led some to advocate lobectomy as the procedure of choice in the treatment of most lung abscesses. Although we were reluctant to accept this radical philosophy, we are now resecting many and draining very few. We have had some excellent results with drainage and believe that it should not be entirely discarded in favor of lung resection. Lobectomy should be reserved for those in which it is apparent that drainage might not cure. Occasionally a pneumonectomy must be done because of difficulties encountered while doing a proposed lobectomy; these incidents are frequent enough to warrant care in selecting resection. Lobectomy should not be chosen merely because it appears to be a more expedient procedure and a means of avoiding a prolonged period of messy dressings. If resection is ever justified solely as an aid to avoid dressings it is in children. The postoperative dressing program of a drained lung abscess is so difficult and the end results of drainage are so poor in children that we have chosen resection in many children who probably could have been cured by simple drainage.

In multilocular abscesses resection is better than multiple attempts at drainage. Chronic fibrotic abscesses should be resected.

TABLE 3

Incidence of Lung Abscesses

	1943	1944	1945	1946	1947	1948-49	Totals
Non Surgical	10	16	9	10	10	15	70
Drainage	43	31	18	13	10	4	119
Resection	1	4	12	13	8	20	58
TOTALS	54	51	39	36	28	39	247

If there is any possibility of bronchogenic carcinoma exploratory thoracotomy and resection are preferable to drainage. Such cases, usually in the older age group, often have some atelectasis associated with the abscess. Routine bronchoscopy of all abscesses will discover some of them.

When we analyze the procedures used in the present series of cases, it is apparent that we are now doing few pneumonostomies and that the ratio of resections to drainages has been completely reversed within the six year period studied. Even if this is a commendable reversal, we admit it is not due entirely to a change in our criteria for electing resection. It is one of the effects of universal antibiotic therapy. The cases we now have referred for surgery are not the toxic, foul, fulminating abscesses of a few years ago. Most of the recent cases have remained on the medical service during the optimal period for drainage, and the surgeon has no choice of methods by the time he is called to see the patient. This trend has proved to be beneficial, for the long-term outlook for pulmonary resection is far better than that of drainage. Besides the obvious advantage of immediately eliminating the disease and shortening the convalescence, resection also eliminates the dangers of hemorrhage, embolic brain abscesses, and putrid empyema. The danger in continuing antibiotic therapy until the abscess is definitely chronic and then resecting the lobe lies in the possibility of obscuring a carcinoma by prolonged chemotherapy. This danger is lessened if every case of lung abscess has at least one bronchoscopic examination early in the course of treatment. Not only will this help to discover unsuspected tumors, polyps, and foreign bodies, but it will also serve to bring a surgical consultant into the case, letting the surgeon share some of the decisions to be made as the abscess develops.

SUMMARY

A series of 247 lung abscesses has been presented, covering a six year period. Of these 188 cases were cured, there being from six months to six years follow-up on most of the patients. Thirty-four patients died (13.7 per cent).

All cases had chemotherapy and bronchoscopy.

The management of lung abscesses depends on the extent of the disease when it is recognized and treatment is begun.

1) In the early formative phase most acute aerobic abscesses can be cured by intensive antibiotic therapy and bronchoscopy. Seventy of the 247 cases presented were treated only in this way.

2) An acute unilocular abscess which does not respond to chemotherapy should have surgical drainage as soon as it is apparent

that more conservative measures are inadequate. One hundred nineteen patients in this series had surgical drainage.

3) Multilocular and chronic abscesses need pulmonary resection. Drainage is inadequate in these cases. Pulmonary resection was done in 58 of the present series.

Although the worth of abscess drainage is appreciated, there is a tendency to restrict its use in favor of more prolonged antibiotic conservatism and lobectomy when this fails. As a result most abscesses are complicated and require resection when the surgeon sees them. Of the 39 cases occurring since January 1, 1948 there were only four drainages and 20 resections; two of the resections were on patients previously considered as cured by drainage.

All patients with lung abscess should have a bronchoscopic examination. Unsuspected foreign bodies or bronchial tumors may be discovered by such routine examinations and the thoracic surgeon is introduced to the case early enough to share in the decisions of treatment.

RESUMEN

Se han presentado una serie de 247 casos de absceso de pulmón, cubriendo un período de seis años. Se curaron 188, la majoria de los enfermos han sido seguidos seis años. Treinta y cuatro enfermos se murieron (13.7 por ciento).

A todos los enfermos se los sometió a la broncoscopia y quimoterapia.

El tratamiento del absceso de pulmón, depende de la extensión del proceso, cuando se hace el diagnóstico.

1) Casi todos los abscesos agudos y aerobios, en la face inicial se pueden curar con el tratamiento de antibióticos y broncoscopia. Setenta de los 247 casos presentados, han sido tratados de esa manera.

2) Un absceso agudo localizado, que no responde a la quimoterapia, debe ser drenado quirúrgicamente, tan pronto como se note, que el tratamiento conservativo es inadecuado. Ciento diez y nueve enfermos en este grupo, han sido drenados quirúrgicamente.

3) Abscesos crónicos multiloculares requieren extirpación. El drenaje es inadecuado en estos casos. La resección pulmonar se ha empleado en 58 casos en esta serie.

Aunque el drenaje del absceso es apreciado, hay una tendencia hacia el uso prolongado de antibióticos y cuando estos fallan se hace una resección pulmonar. Como resultado, cuando la mayoria

de estos enfermos llegan al cirujano, requieren un tratamiento quirúrgico. De los treinta y nueve casos que se presentaron desde el primer de Enero de 1948, solamente cuatro han sido drenados y veinte fueron resecados. Dos de los resecciones eran enfermos que se consideraron curados con el drenaje.

Todos los pacientes con absceso de pulmón deben ser sometidos a la broncoscopia. Tumores y cuerpos extraños serán descubiertos con estos examenes, y el cirujano torácico tendrá toda la información necesaria para llegar al tratamiento.

Discussion

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Dr. Klepser has ably presented and surveyed an excellent part of his work relating to the serious disease, lung abscess.

In a survey of 122 patients with lung abscess at the University Hospital covering a 10 year period ending in 1942, it was evident that there were several major causes for the disease, foreign body, aspiration of secretion at the time of or immediately after operation, infected emboli, and spontaneous abscess; that is, one with no demonstrable cause. As described by Dr. Klepser, removal of the foreign body, if it were the cause usually cured the abscess. Lung abscess from foreign body seemed the most benign. The spontaneous abscess was the most devastating and before use of chemotherapeutic agents this type often promptly caused death.

In rarely having the opportunity to see or treat the patient with early acute uncomplicated abscess, I have much the same experience as Dr. Klepser. The present day chemotherapeutic agents administered adequately have changed the course of lung abscess. A large percentage of patients with lung abscess will recover on medical treatment so long as inspissatated debris does not remain in the abscess cavity. If the abscess is not cured it will be kept localized by medical treatment. The abscess often can be removed by a segmental resection of the lung where as formerly it was often necessary to remove the whole lung if the abscess were to be excised.

Since lung abscess is often a chronic disease the demonstration of the presence or absence of associated bronchogenic carcinoma in the older patient is most difficult even when bronchoscopy, bronchogram and cytologic studies are adequately used. The differentiation from tuberculous cavitation in the younger patient

sometimes is difficult and in all cases a careful search for tubercle bacilli must be diligently made even though the disease is thought to be a simple pyogenic abscess of the lung.

The large percentage cured by chemotherapy and other medical measures and the confinement of the abscess to a single segment or lobe of the lung has brought about the trend toward primary resection as described by Dr. Klepser rather than drainage. The fear of associated bronchogenic carcinoma and the willingness to resect tuberculous lesions of the lung has contributed to the trend toward resection as the surgical treatment of choice for pyogenic abscess of the lung.

I wish to congratulate Dr. Klepser on his fine work and especially his low mortality rate.

Electrocardiographic Changes in Pulmonary Collapse: Artificial and Spontaneous Left-Sided Pneumothorax Studied by Conventional and Unipolar Methods

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In a previous paper Feldman and Silverberg[1] described the electrocardiographic changes produced by the induction of artificial pneumothorax in patients with pulmonary tuberculosis, using Leads I, II, III, and CF precordial leads. A characteristic electrocardiographic pattern was noted in cases with left pneumothorax, consisting of a low T_1, T_3 higher than T_1, together with lowering and frequent inversion of QRS complexes and T waves in CF leads, maximal in the more lateral precordial leads. In several left pneumothorax cases QS complexes were noted in CF_3. The above described changes tended to revert toward normal with tracings taken in the sitting position. The electrocardiographic changes were ascribed to positional changes of the heart together with the interposition of a non-conductor between the heart and the chest wall. The present study was undertaken to determine whether "unipolar" electrocardiography might aid in explaining the changes previously described.

Method

Two categories of patients were studied: Group I comprising those with spontaneous left-sided pneumothorax, Group II composed of patients with pulmonary tuberculosis in whom therapeutic left-sided pneumothorax was planned. In the first group, electrocardiograms were taken while the lung was partially collapsed and repeated when re-expansion had taken place. In the second group, tracings were taken prior to induction of pneumothorax, and then repeated soon after pneumothorax was estab-

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lished. In each case the degree of collapse or re-expansion of the lung was correlated with the electrocardiograms, although previous studies indicated that the degree of pulmonary collapse had little bearing on the electrocardiographic changes. Electrocardiograms were made in both the supine and sitting positions, since the recumbent CF changes in left pneumothorax have been noted to revert toward normal in the upright position.[1,2] In addition to Leads I, II, and III, Leads CF_1 through CF_6, Leads V_1 through V_6 (Wilson terminal[3]), and the augmented unipolar limb leads (Goldberger[4]) were taken. Measurements of the direction and amplitude of the various complexes in each lead were made, and the numerical value of the mean electrical axis of the QRS complexes determined by the triaxial reference system of Bayley. The electrocardiographic position of the heart was estimated from the unipolar limb leads.[5] Chest roentgenograms were taken in the P-A position at a target film distance of six feet in the first and five feet in the second group. Lateral displacement of the heart was determined by measuring the lateral shift of the left contour of the heart shadow at the level of the superior border of the tenth costovertebral junction. The degree of pulmonary collapse was estimated from the chest films.

Results

In Group I, comprising four patients with spontaneous left-sided pneumothorax, the electrocardiographic pattern in Leads I, II, III, and CF_1 through CF_6 in the recumbent position corresponded with the previously described pattern. Since the more pronounced precordial electrocardiographic changes occur toward the lateral portion of the left side of the chest, only positions C_2, C_4, C_5, and C_6 are illustrated. The V precordial leads show a striking and significant difference from the CF leads. In those positions where the CF lead shows a small inverted QRS, or even a QS complex, and inverted T waves, the V lead has an upright QRS and T wave of normal contour, but perhaps of decreased amplitude. These differences are striking in CF_5 and CF_6 as contrasted with V_5 and V_6, while the differences between CF_2 and V_2 are not significant. Marked differences between CF and V leads have recently been described in individuals without evidence of heart disease.[6] The differences in left-sided pneumothorax are illustrated in Figure 1, patient G.C., columns 1 and 3. The conventional limb leads show a low QRS in Lead I, a higher QRS in Lead III, and T_3 larger than T_1. In the augmented unipolar limb leads both the right and left arm tracings reveal the cavitary type of potential, and the left leg a left ventricular epicardial pattern, indicative of a vertical position. On assumption of the sitting position, the CF leads return

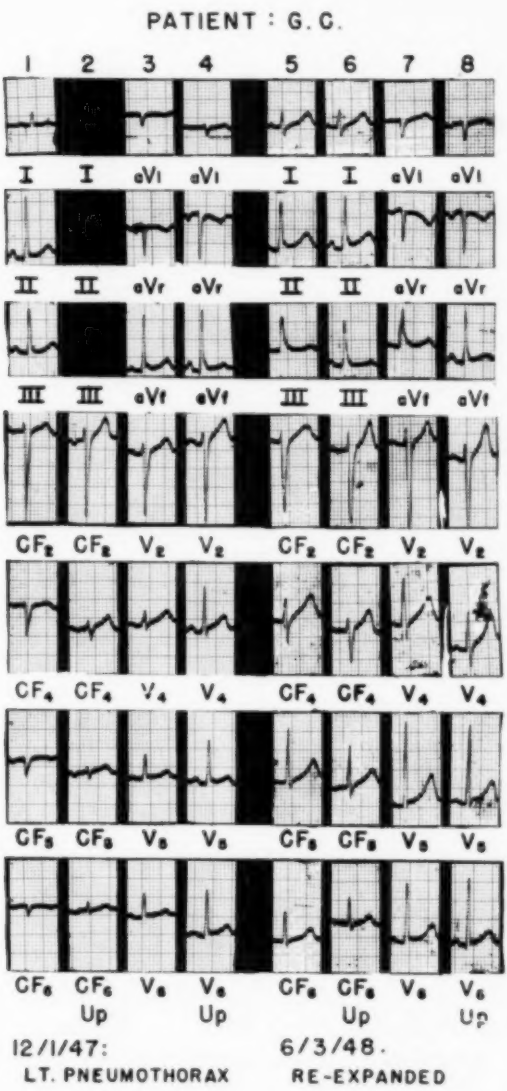

FIGURE 1: Single complexes from each lead in illustrative case of spontaneous left pneumothorax. Columns 1, 3, 5, and 7 were taken in the recumbent position; 2, 4, 6, and 8 in the sitting position (marked "Up"). One to 4 were taken when the lung was approximately 50 per cent collapsed, 5 to 8 after re-expansion.

toward normal, and although the amplitude may be relatively small in CF_1 through CF_6, definite R waves and upright T waves are present in all CF leads. The amplitude of QRS and T waves in the V leads also increases when the patient assumes the sitting position. Although Leads I, II, and III were not recorded in the sitting position in patient G.C., the unipolar limb leads indicate that the heart is less vertical in the sitting position than in the recumbent. Measurements of the mean electrical axes revealed that the heart was less vertical in the sitting than in the recumbent position in four of the five remaining patients. After re-expansion of the lung in patient G.C., Leads I, II, and III are within normal limits with T_1 greater than T_3. The CF and V leads are within normal limits. The unipolar extremity leads indicate a vertical heart, with aV/R representing the cavitary potential, aV/L the epicardial surface of the right ventricle, and aV/F the epicardial surface of the left ventricle. The remaining three patients with a spontaneous left-sided pneumothorax showed similar changes.

In Group II, composed of two patients with pulmonary tuberculosis, the control electrocardiograms were within normal limits. Both hearts were in a semi-vertical position electrocardiographically. The electrocardiograms obtained in patient B.N. before and following the institution of left pneumothorax are illustrated in Figure 2. Prior to the induction of pneumothorax, the electrocardiogram indicated no significant positional change between the sitting and the recumbent position. With pneumothorax present, the heart was more vertical in the recumbent than in the sitting position. The same general pattern occurred in the CF leads as previously described. Precordial V leads, on the other hand, showed a normal contour, although a decreased amplitude. In Figure 2, Lead aV/F taken in the sitting position has an inverted T wave which accounts for the diphasic T_2 and inverted T_3. Similar electrocardiographic changes occurred in patient C.W., who also was subjected to therapeutic left pneumothorax.

Discussion

In a previous paper,[1] a characteristic electrocardiographic pattern was described in patients with therapeutic left-sided pneumothorax, using Leads I, II, III, and CF precordial leads. This pattern was of such a nature that it might give rise to a mistaken electrocardiographic diagnosis of coronary disease. The present study was undertaken to determine whether "unipolar" electrocardiograms might explain the previously described abnormalities.

Leads I, II, III, and CF are related to the V leads as follows:[7a,7b]

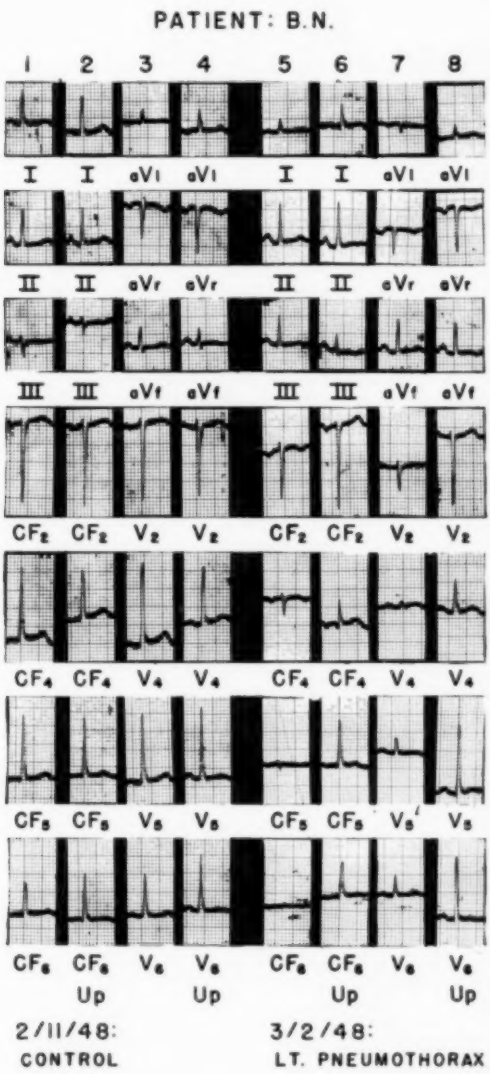

FIGURE 2: Single complexes from each lead in illustrative case of induced left pneumothorax. Columns 1 to 4 taken prior to induction of pneumothorax; 5 to 8 after establishment of pneumothorax with approximately 15 per cent collapse.

TABLE 1: Data in Six Cases of Left-Sided Pneumothorax

	Patient	Age & Sex	Date	Heart Position	Pulmonary Collapse	— — AXIS — — Recumbent	Sitting
					Per cent		
GROUP I	G.C.	26 M	12-1-47	11.0 cm.	50	+80°	
			6-3-48	7.0 cm.	0	+83°	+85°
	J.D.	22 M	12-19-47	6.8 cm.	75	+75°	+65°
			2-20-48	7.0 cm.	0	+36°	+35°
	W.P.	39 M	5-13-48	14.0 cm.	75	+3°	+60°
			7-6-48	13.8 cm.	0	+5°	+13°
	S.S.	24 M	3-22-48	8.5 cm.	95	+80°	+67°
			5-20-48	9.0 cm.	0	+78°	+73°
GROUP II	B.N.	45 F	1-26-48	7.4 cm.	0	+10°	+18°
			3-2-48	7.9 cm.	15	+74°	+52°
	C.W.	45 M	2-5-48	6.2 cm.	0	+58°	+40°
			3-2-48	9.3 cm.	30	+78°	+42°

TABLE 1: Table showing heart position, degree of pulmonary collapse, and electrical axis of heart as determined by the triaxial reference system of Bayley (normal axis deviation is between 0 and +90 degrees; right axis deviation is considered present when the electrical axis is more positive than +90 degrees, left axis deviation when the axis is more negative than 0 degrees). Heart position measured as described in text using chest roentgenograms taken in upright position.

Lead I = Lead V/L − V/R = (aV/L − aV/R) 2/3
Lead II = Lead V/F − Lead V/R = (aV/F − aV/R) 2/3
Lead III = Lead V/F − Lead V/L = (aV/F − aV/L) 2/3
Lead CF = Unipolar (V) precordial lead − Lead V/F = V − 2/3 aV/F

With these relationships in mind, the electrocardiographic changes noted in Leads I, II, III, and CF precordial leads can be explained. The tracings in aV/L, aV/R, and aV/F taken during the pneumothorax explain the low T_1 and the T_3 larger than T_1, on the basis of the markedly vertical heart position. In the presence of left-sided pneumothorax, the amplitude of the unipolar precordial potential is markedly reduced. Since the unipolar left leg potential is greater than that of the unipolar precordial lead, the subtraction of the former from the latter, which occurs in the CF leads, explains the inverted rS or QS complexes, and low or inverted T waves in leads CF_4 through CF_6.

The facts that the potentials in the V leads increase, and CF leads return toward normal and aV/F remains relatively unchanged when the patient is upright, suggest that most of the change is due to the increased distance of the heart from the chest wall with the interposition of a non-conductor (air) between the heart and the chest wall.

In five of the six patients, numerical measurements of the electrical axis and positional interpretation of the unipolar extremity leads indicate that in the presence of left pneumothorax the heart is more vertical when the patient is recumbent than when he is sitting, contrary to the usual findings in patients without left pneumothorax. The probable explanation for this finding is that the pleural air tends to accumulate in the apical portions of the pleural space when the patient is sitting, thus permitting the lower part of the lung to approach the lateral chest wall more closely than in the recumbent position, and allowing the heart to assume a less vertical position.

SUMMARY AND CONCLUSIONS

1) Conventional and unipolar limb leads, and CF and unipolar chest lead electrocardiograms, and chest films were taken in four patients with spontaneous left-sided pneumothorax, and in two tuberculous patients in whom therapeutic pneumothorax was planned. In the first group the studies were made during pneumothorax and following the re-expansion of the lung. In the latter group the studies were made before and after institution of pneumothorax.

2) The characteristic electrocardiographic pattern for Leads I, II, III, and the CF precordial leads was repeated in all six cases. This pattern consisted of a small T_1, a T_3 larger than T_1, QRS

small and inverted (or a QS complex), and low or inverted T waves in the chest leads, the chest lead changes being maximal in the more lateral precordial positions.

3) This previously described pattern can be explained on the basis of the "unipolar" lead findings. The standard limb lead changes are apparently produced by the heart assuming an extremely vertical position in the presence of left pneumothorax. The CF lead abnormalities in the recumbent position are related chiefly to the decreased voltage of the precordial V leads, resulting from the interposition of a non-conductor (air) between the heart and the chest wall.

4) The use of the CF precordial leads in the presence of left-sided pneumothorax might lead to a false electrocardiographic impression of coronary artery disease, or even of old myocardial infarction. In the same situation the V leads (Wilson terminal) show a tracing with a low amplitude but with a more normal contour. This would minimize the likelihood of erroneous interpretation.

SUMARIO Y CONCLUSIONES

1) Se tomaron electrocardiogramas ordinario unipolar con electrodo en los miembros y CF, unipolar con electrodo e nel torax, y radiografias del torax en cuatro enfermos con pneumotórax espontaneo izquierdo, y en dos enfermos en los cuales se habia contemplado el pneumotórax terapéutico. En el primer grupo los estudios se hicieron durante el pneumotórax y después de que el pulmón se ha expandido. En el segundo grupo los estudios se hicieron antes y después de que el pneumotorax se ha iniciado.

2) El tipo de electrocardiograma característico, se repitió en el electrodo I, II, III y en el electrodo precordial en los seis casos. El tipo hallado fué una pequeña T_1, una T_3 mas grande que T_1, QRS mas pequeña e invertida (o el complejo QS) y baja ó onda T invertida, en el electrodo del tórax, especialmente en la posición lateral.

3) Lo descrito previamente, se puede explicar con la idea del electrodo "unipolar." Los cambios obtenidos en los electrodos de los miembros son producidos aparentemente por la posición vertical del corazón, debida al pneumotórax izquierdo. Las anormalidades en el electrodo CF, en la posición horizontal son debidas principalmente a la reducción del voltaje del electrodo precordial, debido a la interposición de aire entre el corazón y la pared costal.

4) El uso del electrodo precordial en los casos con pneumotorax izquierdo puede dar un electrocardiograma falso, por ejemplo: enfermedad de las arterias coronarias ó un infarto del miocardio. Al mismo tiempo el electrodo V (la terminal de Wilson) muestra

un trazado con baja amplitud, pero con un contorno mas normal. Esto contribuirá a la reducción de interpretaciones erroneas.

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Treatment of Bronchogenic Carcinoma with Nitrogen Mustard*

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There have been numerous reports as to the clinical results obtained with the chloroethylamines (nitrogen mustards) in the treatment of Hodgkin's disease, lymphosarcoma and leukemia.[1-11,19] There has been a paucity of reported clinical trials in regard to the treatment of bronchogenic carcinoma with nitrogen mustard despite the fact that a few cases of carcinoma of the lung treated with nitrogen mustard have responded to therapy. Rhoads[12] states that cancer of the lung can be caused to regress temporarily in about 50 per cent of the instances and that these effects are transient and incomplete. The largest series of cases has been reported by Boyland, et. al.[13] Their series included 41 histologically proved cases of bronchogenic carcinoma which were given methyl-bis-(B chloroethyl) amine. These cases were unsuitable for other forms of therapy. Symptomatic relief and objective signs of improvement were noted in approximately one-half of them. Karnofsky has reported temporary symptomatic remissions in four patients with anaplastic carcinoma of the lung.[14] Skinner, et. al.[15] reported treating 25 cases of bronchogenic carcinoma with nitrogen mustard and was of the opinion that clinical improvement was noted in about 70 per cent for periods ranging from six weeks up to six months, after which time the drug became ineffective. Ben-Asher[9] published a report on 11 patients with carcinoma of the lung treated with nitrogen mustard, nine of whom did not respond to therapy. Wintrobe and Huguley[10] treated four cases of bronchogenic carcinoma; two of them had a fair response to therapy. Gellhorn and Jones state in their review, Chemotherapy of Malignant Diseases,[11] that some favorable therapeutic responses have been observed using nitrogen mustard in the treatment of anaplastic carcinoma of the lung, the exact significance of which must remain unsettled until further clinical trials can be undertaken.

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Materials and Methods

In our study we used methyl-bis-(B-chloroethyl) amine. The history, chemistry and pharmacodynamics of the drug have been discussed in detail by others.[16-18] The clinical evaluation of each patient prior to therapy included a careful history, physical examination, evaluation of previous therapy, and confirmation biopsies.

Roentgenographic studies of the chest were made on all patients, and studies of skeletal areas were made when indicated. Studies of the peripheral blood routinely included: hemoglobin determination, red blood cell count, white blood cell count, platelet count and a differential leucocyte count. Other laboratory examinations were done when they appeared indicated.

The nitrogen mustard was given intravenously through the rubber tubing of an intravenous drip of normal saline solution. A course consisted of four injections of the drug. A daily dose of 0.10 mg. per kilogram of body weight was administered.

A hematological study, such as was obtained prior to therapy, was performed every second day during treatment and usually twice a week thereafter. The patient was seen daily and examined at least weekly. Roentgen studies were made as deemed necessary.

Results

A total of 19 male patients were treated with nitrogen mustard. The pertinent data of each patient treated are tabluated in Table 1. We have attempted to evaluate the effect of HN2 (nitrogen mustard) as good, fair or poor. The response has been classified as good when the patient was kept comfortable and free of incapacitating symptoms for six months or more. When the patient had comparable improvement for a shorter period or had fair symptomatic improvement for a longer period (approximately six months), or had marked relief of a serious symptom even though the course of the disease was not altered, the response has been termed fair. When improvement was short-lived or slight, the response has been classified as poor.

The average age of the patients in this series of cases was 54.42 years, the range being from 29 to 68 years. The duration of symptoms prior to HN2 averaged 7.47 months, the shortest time interval being one month and the longest 13 months. Seven of the 19 patients had an exploratory thoracotomy prior to HN2 therapy and were inoperable, three of the 19 had x-ray therapy with poor results. One patient had both x-ray and thoracotomy before treatment was initiated. Ten of the 19 patients had no treatment before HN2. A summary of the results of treatment is given in Table II. Five of the seven patients still living have anaplastic carcinoma and two have squamous cell carcinoma.

TABLE 1

Case No.	Sex and Age	Duration Prior to HN2	Previous Therapy	Effect	Condition Prior to HN2	No. of Courses	Total Dose Mg.	Effect	Other Therapy	Time Since 1st HN2	Present Condition and Comment	Final Evaluation
1	M* 62	8	Surgery	Inop.	Fair	1	17.2-	Fair	None	8 mos.	Living	Fair
2	M 54	13	Surgery	Inop.	Very poor	1	25.2	Poor	None	1 wk.	Dead	Poor
3	M 54	12	Surgery	Inop.	Poor	3½	97.4	Good	X-ray	9 mos.	Living	Good
4	M* 55	10	None		Very poor	1	24.0	Poor	None	1 mo.	Dead	Poor
5	M 49	10	None		Fair	1	25.6	Poor	None	5 mos.	Dead	Poor
6	M 56	4	X-ray	Poor	Poor	1	21.8	Poor	None	1 mo.	Dead	Poor
7	M 56	6	None		Poor	1	26.0	Poor	None	3 wks.	Dead	Poor
8	M 57	9	Surgery	Inop.	Poor	1	29.6	Poor	None	1 mo.	Dead	Poor
9	M 52	11	Surgery	Inop.	Poor	1	25.6	Poor	None	5 wks.	Dead	Poor

Case No.	Sex and Age	Duration Prior to HN2	Previous Therapy	Effect	Condition Prior to HN2	No. of Courses	Total Dose Mg.	Effect	Other Therapy	Time Since 1st HN2	Present Condition and Comment	Final Evaluation
10	M 67	8	None		Poor	1	23.2	Fair	None	3 mos.	Living	Fair
11	M 60	7	None		Very poor	2	46.4	Good	None	6 mos.	Living	Good
12	M 33	8	X-ray	None	Fair	2	58	Poor	None	2 mos.	Dead	Poor
13	M 60	4	None		Very poor	1	21.7	Poor	None	1 mo.	Dead	Poor
14	M 68	6?	None		Fair	1	18.0	Poor	None	1 mo.	Dead	Poor
15	M 62	10	Surgery X-ray	Inop.	Poor	1	20.8	Poor	None	2 mos.	Living	Poor
16	M 53	1	None		Very poor	1	24	Poor	None	4 days	Dead	Poor
17	M 60	5	Surgery	Inop.	Poor	1	24.8	Poor	None	1 mo.	Dead	Poor
18	M 29	2	None		Fair	1	32	Poor	None		Living	
19	M 57	6	None		Good	1	32		None		Living	

*Negro.

The toxic effects of nitrogen mustard when used as a chemotherapeutic agent has been frequently and adequately emphasized by others.[2-7,11,17-19] In this series of cases similar toxic effects were noted. Nausea and vomiting occurred in a high percentage of the cases within one to eight hours following therapy. Nitrogen mustard therapy often resulted in a decrease in the leucocyte count and platelet count, but in no patient did serious complications arise as the result of these effects.

CASE REPORTS

Case 3: K.F., male, age 53 years. *History:* This patient was admitted to the hospital for the first time on July 29, 1948 with the history of frequent attacks of dyspnea for one year prior to entry. Seven weeks prior to admission he had slight hemoptysis, followed in a few days by hoarseness and severe pain in the right anterior chest at the level of the second interspace and radiating to the sternum. Three weeks prior to entry he

TABLE 2

Summary of Results of Treatment with Nitrogen Mustard

Total Number of cases			19
Male			19
Female			0
Average age (years)			54.42
Range			(29 to 68)
Average duration of disease (months)			7.47
Range			(1 to 13 mos.)
(Prior to HN2)			
Number explored surgically			7
Number previously treated with x-ray			3
Number initially treated with HN2			10
Number of courses given (Range)			(½ to 3½) — (total 23½)
Condition prior to HN2:			
		No. Living	Effect of HN2*
1) Good	1	1	2
2) Fair	5	2	2
3) Poor	8	3	13
4) Very poor	5	1	
*Two patients treated too recently to be evaluated.			
Number patients living since HN2 started			7
Months living since HN2 started			2-9
Number patients dead since HN2 started			12
a) Length of life following HN2			2 days to 5 months
Final evaluations:*			
1) Good	2		
2) Fair	2		
3) Poor	13		
*Two patients treated too recently to be evaluated.			
One patient received x-ray therapy following HN2.			

had an episode of coughing and developed severe pain in the left antecubital fossa which radiated to his left shoulder; within a half hour his left arm began swelling and within two hours it was greatly enlarged. He lost 20 lbs. in two months. Past history revealed that he was discovered to have positive serologic test for syphilis 20 years prior to entry and had received antisyphilitic therapy intermittently for two years.

Physical Examination: Weight 159 lbs., B.P. 120 80 R.A., 130 100 L.A. He appeared chronically ill. His left pupil was larger than the right. The

TABLE 3: Patients Still Alive

Case No.	Type of Carcinoma	Comment (Clinical Improvement
1	Anaplastic (suggestive of epidermoid)	Slight gain in weight.
3	Anaplastic	Edema of face, arms and legs—disappeared with relief of pain following 1st course (relief of caval obstruction). Following 2nd course similar effect.
10	Squamous	Gained 6 lbs. weight, improved appetite. No further hemoptysis, more energy.
11	Undifferentiated type	Gained 21 lbs. weight and energy. Increased appetite.
15	Anaplastic	No improvement. Weight loss.
18	Poorly differentiated Suggestive of epidermoid	Treated too recently to be evaluated.
19	Squamous	Treated too recently to be evaluated.

TABLE 4: Patients Dead

Case No.	Type of Carcinoma	Length of Life Following HN2
2	Epidermoid	1 week
4	Anaplastic	1 month
5	Poorly differentiated epidermoid	5 months
6	Anaplastic	1 month
7	Anaplastic	3 weeks
8	Poorly differentiated epidermoid	1 month
9	Undifferentiated	5 weeks
12	Poorly differentiated epidermoid	2 months
13	Undifferentiated	1 month
14	Anaplastic	1 month
16	Undifferentiated	4 days
17	Epidermoid	1 month

right jugular vein was prominent. There was dulness to percussion over the left base posteriorly with decreased breath sounds over this same area. Dilated veins were visible over the left antero-thoracic cage and over the left arm. The left arm was edematous from his shoulder to his wrist. His legs and face were also edematous. Venous pressure determinations on August 3, 1948 revealed a V.P. of 375 mm. citrate in the right antecubital vein and 140 mm. citrate in the left. Circulation time with decholin was 26 seconds, with ether 14.5 seconds. Kahn and Wassermann tests were both reported as doubtful.

Roentgenogram and fluoroscopy of chest on July 28, 1948 revealed a soft tissue mass occupying the anterior superior mediastinum and was interpreted as an aneurysm of the ascending arch of the aorta.

Because of the previous history of antisyphilitic therapy, doubtful serologic findings and diagnosis of aortic aneurysm, antiluetic treatment was begun. He received penicillin 5,000 units intramuscularly every three hours for three days and then 50,000 units every three hours for 15 days. He was seen by the consultant in Chest Surgery who recommended exploratory thoracotomy with the consideration of possibly wrapping the aneurysm with polythene cellophane. However on August 21, 1948 he was again fluoroscoped and no pulsation of the mediastinal mass was observed.

Exploratory Thoracotomy was performed on August 23, 1948 and a large inoperable nodular tumor was found in the mediastinum about the great vessels especially beneath the arch of the aorta and carotid. Biopsy was done.

Pathological Report: Anaplastic carcinoma of bronchogenic origin.

Treatment: Nitrogen mustard was administered (7.6 mg. per day) for four days beginning September 4, 1948. By September 11 edema of the legs was greatly improved, and the face and arms were free of edema.

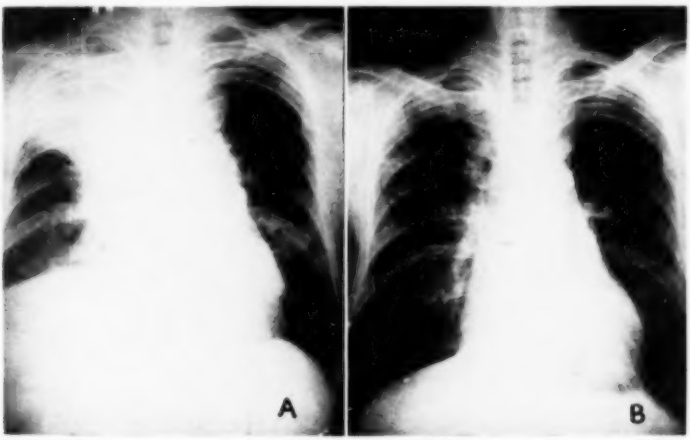

FIGURE 1. Case 3-A, March 9, 1949: Atelectasis right upper lobe with marked widening of the mediastinum.—Case 3-B, April 9, 1949: After HN2 therapy, regression of atelectasis, right upper lobe and diminution of the mediastinal widening. This was associated with marked clinical improvement.

X-ray film of chest on September 17 did not reveal the previously noted soft tissue mass in the anterior mediastinum although there was some increase in density in the area it previously occupied. Pain was relieved by small amounts of codeine and cough and hoarseness following therapy were less. He was discharged from the hospital on October 7, 1948 and was seen several times before further therapy was indicated. He remained comfortable except for occasional epistaxis and dull chest pain which was relieved by codeine and aspirin. Three weeks before readmission or three months after HN2, he again became hoarse, and one week before entry he had severe chest pain, hemoptysis and pedal edema. He was readmitted to the hospital on December 6, 1948 when he weighed 142 lbs. X-ray inspection of chest showed little if any change. He received 6.5 mg. of nitrogen mustard per day for four days beginning December 8th.

The ankle edema again disappeared and there was no further hemoptysis. X-ray film of chest on December 21 revealed no further evidence of a mediastinal mass. He was discharged from the hospital on January 5, 1949 and was seen several times thereafter. During the interval before readmission he was again fairly comfortable; two months before re-entry he began to note dyspnea on exertion (this was one month after second course of nitrogen mustard). However, dyspnea did not become severe until one week before entry. Three days before admission he had ankle edema, hemoptysis, hoarseness and venous distension over chest. He was hospitalized on March 8, 1949 when he weighed 143½ lbs. X-ray inspection of the chest on March 9 revealed a homogenous increase in density in the upper one-half of the right lung field and increase in density in the left suprahilar region. He received 6.5 mg. nitrogen mustard per day for four days beginning March 10.

Dyspnea became greatly improved and he gained two pounds following therapy. His appetite, energy and feeling of well being all increased. X-ray inspection of chest on April 9, 1949 still revealed some widening of the mediastinal shadow but appeared diminished in extent. The previous density in the upper half of the right lung field had completely disappeared. Symptomatic remission was brief however and on April 21 there was considerable swelling of the neck. There was also noted distension of the superficial veins of the neck, chest and abdomen. X-ray film of chest on April 21 revealed widening of the superior mediastinum with possibly slight increase in the width of the mediastinum compared to previous x-ray films. The lungs were still clear.

He was given 6.5 mg. nitrogen mustard per day for two days beginning April 23, 1949. This brief course of therapy was given because of the short interval since his full course of treatment. The effect again was good but transient; because of this and his discouragement, it was felt that a trial of x-ray therapy was indicated. This was instituted on May 11, 1949. The neck vein distension disappeared after four treatments and he was more comfortable. X-ray inspection of chest on June 15, 1949 revealed reduction in the size of the mediastinal mass.

Case 2: S.H., male, age 60 years. *History:* This man was admitted to the hospital on January 6, 1949 with the history of frequent pains in the chest and shoulders since June, 1948. He developed generalized edema and lost 67 lbs. of weight in six months. In October, 1948, he had a productive cough, occasional hemoptysis and became dyspneic. Shortly before entry he began to regurgitate food through his nose and had difficulty in swallowing.

On examination he weighed 123 lbs. B.P. 140/80. He was poorly nourished, appeared acutely ill, spoke hoarsely, and was weak. There was limitation of motion of both sides of the chest, more marked on the right. There was dulness to percussion over the right base with absent breath sounds over this area. The liver was felt two to three fingers breaths beneath the right costal margin and was slightly tender. There were slight edema of legs and slightly enlarged bilateral inguinal and axillary nodes.

X-ray inspection of chest on January 10, 1949 revealed a homogenous increase in density of the right base. An infiltrative process was visualized radiating outward and downward from the right hilum region. Fluoroscopy of the esophagus and chest showed an irregular defect in the cervical portion of the esophagus which also involved the hypopharynx. Puddling in the pyriform sinus indicated esophageal obstruction. On January 21 esophagoscopy was negative, and bronchoscopy showed the presence of a soft nodular mass in the right middle lobe orifice. Biopsy revealed undifferentiated bronchogenic carcinoma.

It was agreed that the bronchogenic carcinoma had probably spread to the mediastinum and had caused obstruction by extrinsic pressure on the esophagus and therefore was inoperable. Therefore HN2 5.6 mg. per day was administered for four days beginning January 26, but he gradually developed further esophageal obstruction and suction was required to remove accumulation of mucus. Cough increased in severity. Dyspnea increased and he became weak and practically bed-ridden. X-ray film of chest on March 31, 1949 showed a moderate increase in the infiltrative process involving the right lower lung field. Swallowed barium failed to reveal any definite evidence or narrowing of the esophagus. His weight was 123 lbs. He received 6.0 mg. HN2 per day for four days beginning March 28, 1949.

His condition was poor at the beginning of nitrogen mustard therapy, but his appetite improved and he gradually gained weight and energy. The dyspnea improved and he was able to swallow solids. He left the hospital on leave on April 15 and felt so improved that he desired to remain at home. He returned on May 12 when he felt extremely well and weighed 135 lbs. X-ray film of chest on May 15 showed the previously reported area of increased density in the region of the right middle lobe. Swallowed barium did not reveal any disease of the esophagus. He was discharged on May 21. On June 21, 1949 he felt considerably stronger, his appetite had remained good and he had gained 3 lbs. in weight in the month following discharge from the hospital. He had not had any further symptom of esophageal obstruction.

SUMMARY

1) The effects of methyl-bis-(B chloroethyl) amine in 19 patients with histologically proved bronchogenic carcinoma are described. These cases were unsuitable for other forms of therapy.

2) Seven of the 19 patients are still alive. Five of the seven living have anaplastic carcinoma.

3) A good effect was obtained in two patients having anaplastic carcinoma. One is still living nine months after three and one-half courses of nitrogen mustard therapy. The other patient is

still living six months following two courses of HN2. Both had superior mediastinal involvement with decrease in tumor size and symptoms following therapy.

4) Nitrogen mustard often resulted in decrease in the leukocyte count, and platelet count, but in no patient did serious complications arise as the result of these effects. Toxic manifestations of nausea and vomiting were observed in several patients following HN2 administration, but the intensity of these symptoms varied greatly.

CONCLUSIONS

Methyl-bis-(B chloroethyl) amine is a useful drug in the treatment of some cases of inoperable bronchogenic carcinoma. This is especially true if the carcinoma is anaplastic. Whether nitrogen mustard is more effective than x-ray therapy in the treatment of bronchogenic carcinoma remains to be evaluated. Ultimately nitrogen mustard therapy, like irradiation, proves ineffective. Ease of administration, availability in communities where irradiation may not be obtainable and absence of skin reaction are advantages of nitrogen mustard as compared to roentgen-ray therapy. Methyl-bis is a useful drug, but is toxic and must be administered with care. Studies must be repeatedly made on the patient's blood to follow the state of the bone marrow.

RESUMEN

1) Se han descripto los efectos de la amino-metil-bis (B cloroetil) en 19 enfermos con cáncer broncogénico provado histológicamente.

2) Siete de los 19 estan todavia vivos. Cinco de los siete vivos tienen cáncer anaplástico.

3) Se ha obtenido un efecto bueno en dos enfermos, que tienen cáncer anaplástico. Un enfermo está todavia vivo, nueve meses después de haber recivido tres series y media de tratamiento con el nitrógeno de mostaza. El otro enfermo también está vivo, seis meses después de recibir dos series de la misma droga. Los dos presentaron invasión del mediastino superior, el cual decreció en tamaño después del tratamiento.

4) El nitrógeno de mostaza generalmente produce una disminución de los glóbulos blancos y plaquetas, pero ningún enfermo ha sufrido serias complicaciones como consecuencia del mismo. Se han observado manifestaciones tóxicas, como ser nauseas y vómitos, en varios enfermos, después de la administración de la droga, la intensidad de estos síntomos, varia grandemente.

CONCLUSIONES

Metil-bis-(B cloroetil) amino es una droga útil, en el tratamiento de algunos casos inoperables de cáncer broncogénico. Esto es especialmente cierto en el caso de cáncer anaplástico. Todavía no se ha podido avaluar si el tratamiento con esta droga, es mas efectivo que el tratamiento con los rayos X, en el cáncer broncogénico. Finalmente, esta droga como los rayos X, no son efectivos. Esta droga presenta algunas ventajas sobre el tratamiento con rayos X, como ser fácil administración, fácil de obtener en lugares en los cuales no hay rayos X y la ausencia de lesiones de la piel. Esta droga es útil, pero es tóxica y debe ser administrada con cuidado. Deben hacerse análisis repetidos de sangre de los enfermos, para seguir de cerca el estado de la médula ósea.

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A Slide Culture Method for Streptomycin Sensitivity Testing*

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Ever since Koch first cultivated the tubercle bacillus, methods have been sought to hasten its growth (in vitro). In his original communication Koch[1] described a technique of examining cultures of tubercle bacilli grown in coagulated blood serum placed in watch glasses or hollow glass slides. He examined these preparations by low-power magnification and was able to detect growth in one week. Pryce[2] in 1941, stimulated interest in a slide-culture method for cultivating tubercle bacilli in sputum. He made slide cells by placing a glass ring over a micro-slide to which a liquid medium was added. With this method colony formation was visible after only three or four days. Rosenberg[3] modified this technique by decontaminating the sputum smears with acid and entirely immersing the slides in tubes of liquid medium. Muller[4] also described a modification of Pryce's technique which he used to estimate the bacteriostatic power of chemicals on the tubercle bacillus. Berry and Lowry[5] reported early growth of tubercle bacilli from pathological material with a similar technique and gave an excellent review of the slide culture literature.

Because the existing methods[6] of performing streptomycin sensitivity tests are laborious and time-consuming and indeed are often too slow to be of clinical value, it was suggested by two groups of workers that the slide culture method could be adapted to rapid streptomycin sensitivity testing. Giammalvo and associates[7] employed a laked-blood medium whereas Cummings and Drummond[8] suggested the use of Tween-albumin medium.[9]

This study concerns itself with a comparison of the slide-culture methods with the solid culture technique used routinely by most laboratories.

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*Investigation begun at the Tuberculosis Evaluation Laboratory, Communicable Disease Center, Public Health Service, Atlanta, Georgia, and completed at the Tuberculosis Research Laboratory, Lawson Veterans Administration Hospital and Department of Medicine, Emory University School of Medicine, Atlanta, Georgia.

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Method

Sputum specimens were obtained from patients prior to, during, and after streptomycin therapy. The specimens were divided; one part was treated by the classical NaOH method of concentration[6] and seeded on tubes of Lowenstein-Jensen medium containing graded amounts of streptomycin (0, 1, 10 and 100 micrograms per ml. of medium). These were incubated at 37 degrees C. and examined at weekly intervals for a total of five weeks. The remaining portion of the sputum was smeared over a series of five microslides (3 x ½ inches) which were decontaminated by immersion in Petri dishes containing 5 per cent H_2SO_4. After 10 minutes the slides were transferred to Petri dishes containing sterile distilled water and left for two minutes. The slides were then removed with sterile forceps and placed in tubes of Dubos medium containing the same concentrations of streptomycin; two slides placed in medium without streptomycin serve as controls. Oval culture tubes (23 x 11.5 mm.; length 152 mm.) were employed for the purpose of allowing direct microscopic examination of the slide through the flat surface of the tube. The slide was introduced in contact with the flat wall of the tube to which it was held by a film of medium drawn up by capillary attraction. The tubes were incubated at 37 degrees C. and examined at the end of five days. If colonies with serpentine cords were noted, the slides were removed and stained by the Ziehl-Neelsen method. If no growth was noted the cultures were reincubated for a total of 14 days at which time all slides were removed and stained. Growth was nearly always detectable by the seventh day.

A record was kept of the time of appearance, number, and size of colonies appearing by both culture methods. The final reading was determined by the least concentration of streptomycin which completely inhibited growth.

Results

In this series of 176 sputum specimens it will be seen (Table 1) that positive cultures were obtained by both methods in 38 per cent of cases. Both cultures were negative in 39.2 per cent of the tests, giving a total agreement of 77.2 per cent for the two methods. The routine culture revealed 34 positive (19.3 per cent) which were missed by the slide-culture method, whereas only six positives (3.5 per cent) obtained by slide-cultures were missed by the routine culture method.

Of the 67 cultures which were positive by both methods, there were disagreements in sensitivity readings in only eight tests. Table 2 reveals that in most of these instances a higher degree

of resistance of tubercle bacilli to streptomycin was obtained by the routine culture method.

Discussion

The slide cultures were examined and could be interpreted two weeks before the routine cultures were positive. This was of great advantage clinically since any change in therapy could be instituted more rapidly. However, in those cases in which the sputum contained only small numbers of tubercle bacilli as evidenced by direct smear examination, the slide culture at times showed no growth. The inability to detect small numbers of tubercle bacilli appears to us to be the greatest limitation to the use of the slide culture method. This is probably due to the small amount of sputum which can be examined by the slide-culture method in comparison to the large amount of material which can be seeded on routine cultures. When pure cultures are employed the slide cul-

TABLE 1

Comparison of Growth on Slide Culture and Routine Culture Streptomycin Sensitivity Tests Performed Directly from Sputum Specimens

	Number	Per cent
Slide culture +, Routine culture +	67	38.0
Slide culture −, Routine culture −	69	39.2
Slide culture +, Routine culture −	6	3.5
Slide culture −, Routine culture +	34	19.3
Totals	176	100.0

TABLE 2

Comparison of Streptomycin Sensitivity Readings on Tubercle Bacilli Obtained by Slide and Routine Culture from 67 Specimens in Which Both Methods Yielded Growth.

	Number	Per cent
Slide culture sensitive to 10 mcg. ml. Routine culture resistant to 10 mcg. ml.	6	9.0
Slide culture resistant to 10 mcg. ml. Routine culture sensitive to 10 mcg. ml.	2	3.0
Total disagreements	8	12.0
Slide and routine readings same *Total agreements*	59	88.0
Total tests	67	100.0

ture technique supports the growth of small numbers of tubercle bacilli.

It is interesting to note that from a study of the size of the micro-colonies one can estimate "partially resistant strains" by comparing colony size on the control slide with the size of the colonies on the slides grown in streptomycin containing medium. Another advantage of the slide culture method is that the relatively short period of incubation does not allow for the development of resistance of the tubercle bacilli *in vitro*, a possibility which as yet has not been ruled out in the routine culture method which requires prolonged incubation.

SUMMARY

1) A rapid slide culture method for performing streptomycin sensitivity tests is described.

2) A comparison of this technique with the routine streptomycin sensitivity method is made on 176 sputum specimens.

3) The slide-culture technique has the advantages of simplicity and rapidity but at times failed to show growth when small numbers of tubercle bacilli were present in the specimen.

RESUMEN

1) Se describe un método rápido de porta-cultura para controlar la sensitividad a la streptomicina.

2) Se compara esta técnica, con la técnica ordinaria para la sensitividad a la streptomicina en 176 esputos.

3) La técnica del porta-cultura, tiene la ventaja de la rapidez y simplicidad, pero a veces no muestra crecimiento, cuando el número de bacilos tuberculosos es pequeño.

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Principles and Practices in Lobectomy (Including Segmental Resection) and Total Pneumonectomy for Bronchiectasis and Chronic Lung Abscess*

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Intrathoracic surgery for bronchiectasis and chronic lung abscess should never be the last resort in the sense in which that term is usually used medically. The results will never be as good as they can be, in respect to either life or health, if the surgeon sees the patient only after he has become a chronic invalid, after medical measures have been tried futilely and ineffectively over long periods of time, and when surgery is literally all that lies between him and death or a life of invalidism. On the other hand, surgery *should* be the last resort for these patients in the sense that an intrathoracic operation is always a serious procedure, which should never be undertaken lightly and unadvisedly, but only after due consideration of all the circumstances and of all possible therapeutic procedures, and only after the risk inevitable in all such operations has been weighed against the risk of permitting a progressive disease to continue unchecked.

Bronchiectasis and lung abscess are diseases which the surgeon practically never sees except by referral, though they are both conditions which ought to be managed from onset to conclusion in close cooperation with the internist. Ideally, the surgeon would see patients with these diseases almost as soon as the medical man sees them, so that he might follow the progress of the case and determine whether surgery is needed at all and when would be the optimum time to perform it. That does not usually happen, but certainly, when the patient is once referred to him, the surgeon cannot manage him without the assistance of the internist.

Bronchiectasis

The first lobectomy for bronchiectasis[1] was performed by Heidenhain in 1901 but for technical and other reasons, including the absence of preoperative preparation and postoperative care as we understand them today, the mortality was high, many operations could not be completed, and patients who survived surgery

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often remained uncured. The tide did not begin to turn until 1929, when Brunn[2] reported the performance of six one-stage lobectomies, with only one death, an unparalleled record up to that time. Three years later Shenstone and Janes[3] introduced tourniquet resection, now regarded as a crude method but then extremely useful, and one technical refinement after another followed. Between 1901 and 1925 only 85 lobectomies for bronchiectasis were recorded in the literature. Between 1925 and 1934 an additional 127 were performed,[1] and series far exceeding that number are now being reported by individual surgeons and groups of surgeons.

When I was a medical student, interne and surgical resident, the surgeon who undertook lobectomy and pneumonectomy was regarded as a daring man, too daring indeed, in the opinion of some critics. It would probably be just as well if we continued to view these operations with a wholesome respect. Because of their lethal potentialities one must be certain that when they are advised they are fully justified and that the risk which they inevitably carry is more than compensated for by the risk undergone by the patient who continues to live with his disease and is not submitted to surgery.

Mortality

A prohibitively high mortality formerly accompanied lobectomy. It was 60 per cent in the first cases reported by Lilienthal[4] in 1922 and 34 per cent in the 212 cases reviewed by Graham, Singer and Ballon[1] in 1934. But only five years later, Edwards[5] was able to report for the preceding 10-year period 168 lobectomies with a total mortality of 12 per cent and with a mortality of 3.7 per cent for the last 54 cases. In the same year Churchill and Belsey[6] reported 124 cases with a mortality of 2.4 per cent, and even more remarkable is the recent report by Meade, Kay and Hughes,[7] from the Kennedy General Hospital Thoracic Surgery Center, of 236 lobectomies, the great majority for bronchiectasis, with only 1 death in the series.

In this same connection it is well to remember Graham's[8] brilliant demonstration of how little lung tissue one may exist with, and how much one may live without. In 1940 he reported the history of a 14-year-old boy, who had been a practical invalid from bronchiectasis since he had had pneumonia and empyema at the age of 2½ years. In a two-stage operation Graham removed all the pulmonary tissue on both sides except the upper lobes and thus transformed the unfortunate child into a healthy, completely normal boy.

Against these statistics of what can be achieved by surgery in bronchiectasis must be set the picture of the untreated or medically

treated disease. As Alexander[9] forthrightly says, the prognosis is very much worse than those who have not investigated the matter are willing to admit. Unfortunately, the figures are incomplete, partly because it is only recently that patients with bronchiectasis have been admitted to hospitals in any considerable numbers and partly because deaths of bronchiectatic patients are usually not assigned to the primary condition but are attributed to pneumonia or even to non-thoracic conditions.

Investigations of small series of cases, however, show that the life expectancy of persons with bronchiectasis is frankly poor. Studies by Perry and King[10] and by Bradshaw, Putney and Clerf[11] show that on the average patients who have had symptoms since early childhood and who are not properly treated do not live much beyond the age of 30 years. In the fatal cases in these series the life expectancy after the development of symptoms was only $13\frac{1}{2}$ years, and the duration of life from diagnosis to death was less than two years.

When one considers the individual patient, the picture is even gloomier. In Riggins'[12] series, patients with bronchiectasis were more frequently admitted and re-admitted to the hospital, and had more frequent respiratory infections, especially bronchopneumonia, than even patients with tuberculosis.

Alexander[9] gives an impressive list of the complications to which patients with bronchiectasis are susceptible: repeated attacks of acute suppurative pneumonitis; pulmonary abscess and gangrene, though these are surprisingly infrequent; septicemia; pleural empyema; spontaneous pneumothorax; hemoptysis; pulmonary fibrosis; emphysema; cor pulmonale; myocardial degeneration; cardiac decompensation; nephritis and amyloid disease; suppurative pericarditis; arthritis; carcinoma from metaplasia of the chronically inflamed bronchial mucosa; and cerebral abscess and meningitis, to which patients with pulmonary suppuration are peculiarly susceptible.

Then the psychologic aspects of the disease must be considered. A foul breath and an uncontrollable productive cough make the patient socially and economically unacceptable. Secondary respiratory complications limit his productive capacity, so that unemployment and poverty are frequent. Only 25 per cent of the patients with this disease studied by Riggins[12] at the Lenox Hill Hospital were doing full time work, usually because the psychologic effects of the disease, combined, in a good many cases, with the ease with which relief could be secured, had killed the desire for self-support. Personality problems are frequent and may be extremely serious. Three of the 100 patients in Riggins' series attempted suicide and one developed paranoia.

What all of this amounts to is that the mortality and morbidity of untreated and medically treated bronchiectasis are such that physicians who routinely advise against surgery in operable cases are assuming a great responsibility. My own experience is that they are tending more and more to assume the responsibility of recommending surgery in such cases, but Churchill[13] is still correct when he calls attention to "the patient who returns to his family doctor for advice and is told that the operation (lobectomy) is an impossible one and that to consent to it means certain death."

Indications for Surgery

The most important of the indications for surgery in bronchiectasis is the irreversible character of the disease. It is true that the character of the dilatations do not influence symptoms, prognosis or treatment and that the extent of the disease does not necessarily determine the need for surgery. Whether a patient with well developed lesions has minimum symptoms or a patient with a few bronchiectatic sacculations has disabling symptoms depends upon whether drainage is free or is almost completely blocked. None of these considerations, however, alter the fundamental fact that when once fibrous replacement of the diseased bronchopulmonary tissue has occurred, there can be no regeneration of the destroyed elements in the bronchial wall. If the irreversibility of the disease is borne in mind, it will be obvious why most medical measures, including bronchoscopy, postural drainage, and chemotherapy and antibiotic therapy are usually only palliative.

Another point to be borne in mind in evaluating the indications for surgery in any given case of bronchiectasis is that the status of the patient at the time of examination must not influence the physician's decision. Bronchiectasis is a disease of recurrences. A patient who is quite well one month may the following month, or the following week, have an exsanguinating series of hemoptyses or a fatal attack of pneumonia. It is not the patient's current status which should determine his treatment, but the recognition of the hazards that may face him in the future.

Except for patients with minimal disease, for whom observation is sufficient, combined, as necessary, with medical measures, all persons with bronchiectasis who can be submitted to surgery should be. It is unfortunately true, however, that when bronchiectasis is extensive, as it is in about half the patients seen, lobectomy is contraindicated, and even pneumonectomy may not be possible. In view of these facts, how is the patient with bronchiectasis who is suitable for surgery to be selected?

First, his age must be considered. Age, per se, is not a contra-

indication to surgery, though the older the patient, the greater the risk. In Edwards'[15] series, which is typical of others, there were no deaths in the 38 patients between four and 16 years of age, but the mortality in the fifth decade was 31 per cent. There are several reasons for this discrepancy. One is that in persons who are still growing (Fig. 1) a true hyperplasia of the pulmonary tissue left in situ will compensate for the function of the excised segment or lobe, while in adults compensatory dilatation of the alveoli, with emphysema, militates against a good result. Another reason is that in children and young adults the lung fissures are well developed and hilar dissection is simpler, while in older persons inflammatory changes in lymphatic tissue may obscure the anatomic divisions. Incidentally, the reason lobectomy is so seldom performed in patients over 40 or 45 years of age is not only the risk attached to it but the fact that persons with the disease usually do not survive to this period of life unless the lesions are so mild that surgery is not required (Fig. 2).

Second, the patient's condition must be evaluated, particularly the cardiorespiratory functional reserve. Anemia, infections, in-

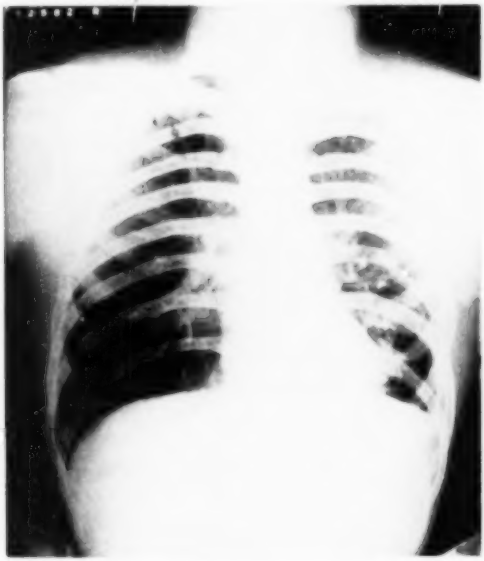

FIGURE 1: Roentgenogram of chest showing expansion of remaining pulmonary tissue following left lower lobectomy for bronchiectasis in 14-year-old boy. The left upper lobe now fills the left hemithorax. Operation converted this boy from a complete invalid to a normal, healthy lad who at the time this picture was taken (six months after operation) was engaging in all forms of athletics.

cluding nasal infections and sinusitis, and similar correctable conditions are not contraindications, but serious cardiovascular and cardiorenal disease would make one hesitate, exactly as they would make one hesitate to perform non-urgent surgery in any other part of the body. Asthmatics, incidentally, are always poor risks.

Third, the ideal that nearly all patients with bronchiectasis should be operated on is severely circumscribed, as Adams[14] points out, by physiologic and pathologic conditions. Lobectomy imposes a heavy tax upon the reserve mechanisms of respiration and circulation. The possibility that these reserves would become greater if what he calls the "fixed charges" against them, in the form of chronic infection, could be removed must be weighed against whether the initial physiologic load imposed by operation could be carried. That is one reason why young persons are better risks than older individuals.

Fourth, the extent of the disease determines the applicability of operation, since the incomplete removal of diseased tissue is unlikely to produce a complete cure. Generally speaking, surgery is particularly applicable to patients whose disease is restricted

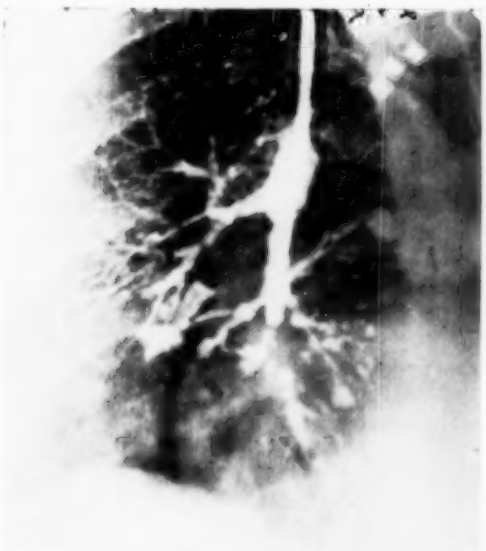

FIGURE 2: Roentgenogram of chest showing tubular and saccular bronchiectasis in right middle and lower lobes in a 46-year-old woman who had had respiratory difficulties since the age of 10. Removal of the affected lobes resulted in complete relief of all symptoms. The pathologist reported the bronchiectasis to be on a basis of congenital cystic disease.

to (a) a single lobe of either lung; (b) the lower and middle lobes on the right or the lower and lingula (middle) lobes on the left; (c) all lobes of one lung; (d) one lobe of each lung; or (e) two lobes of one lung and one lobe of the other lung.[9]

Bilateral bronchiectasis, in the opinion of some observers, is associated with surgical risks which outweigh the possible therapeutic results. This is because for a few weeks after lobectomy the principal respiratory burden is assumed by the opposite, intact lung, and when surgery has been performed on both lungs, that burden may become intolerable. On the other hand, the risk should at least be weighed. Overholt and his associates[15] found that patients with bilateral disease who were submitted to bilateral segmental lobectomy were strikingly better than those who had been treated by unilateral lobectomy. The pulmonary reserve in most cases was practically as good as before operation, which might have been expected, since only functionless parenchyma had been excised and no normal-functioning tissue had been sacrificed.

Preoperative Preparation

Technical improvements have had a great deal to do with the wider employment of lobectomy in bronchiectasis and with the enormous reduction in the morbidity and mortality but the preoperative preparation of the patient, and his postoperative care have also played a major role in these results. Most patients with bronchiectasis are initially poor surgical risks, as might be expected, because of their long-standing suppurative disease. They require the same general preparatory measures for operation which any patient with a chronic infection would require, and they must have, in addition, special local preparation. An operation for bronchiectasis is never an emergency. It may be urgent in a patient with hemoptysis, and it should not be unduly delayed in any patient, but there is never justification for operation without adequate preoperative preparation.

Specifically: 1. A normal plasma protein level must be reestablished. These patients lose great quantities of protein in their secretions, and hypoproteinemia retards wound healing, is a cause of wound rupture, and favors infection. The most satisfactory way to correct this abnormality is to feed the patient a high protein diet. If this measure is inadequate, plasma infusions and the injection of amino acids must be employed.

2. Nutrition must be improved by a high caloric diet, with a high carbohydrate content. Ochsner's[16] suggestion is good, that the administration of thiamine chloride will frequently so stim-

ulate the appetite that the patient is willing to ingest a large amount of food.

3. Vitamin deficiencies must be corrected, particularly vitamin C, which retards wound healing by interference with the normal deposition of collagen. Hypoprothrombinemia is sometimes present in cases of hemoptysis and vitamin K should be administered if there is a prothrombin deficiency.

4. The correction of anemia of any degree is essential. This usually requires repeated transfusions of whole blood, since it is unsafe to undertake thoracic surgery unless the red blood cell count is at least 4,500,000 per cubic millimeter, and preferably higher. Careful typing and investigation of the Rh factor are, of course, routine. Moreover, as Ochsner[16] warns, the normal albumin-globulin ratio must be re-established before the administration of blood is begun. It is frequently perverted in patients with long-standing suppurative disease, who have lost a great deal of protein, and severe transfusion reactions are likely to occur unless this precaution is observed.

5. Antibiotic therapy or chemotherapy is an essential part of the preparation. I prefer to use penicillin in large amounts, at least 25,000 to 50,000 units every three hours intramuscularly for four to seven days before operation, supplemented, in special cases, by aerosol administration. The sulfonamides are substituted for penicillin, or are combined with it, if the response to penicillin alone is unsatisfactory. Postoperative infections are materially decreased by preoperative chemotherapy, and the patient comes to operation with greatly diminished sputum, which is usually no longer purulent.

6. Postural drainage is also an essential part of the preoperative preparation. The optimum position must be determined for each patient, under the supervision of the internist, and definite instructions must be given as to how drainage is to be carried out. It is not an agreeable business, and patients and nurses alike need encouragement to persist in it.

7. Bronchoscopy must be performed routinely before operation, for drainage purposes, though I agree with Mousel[17] that for psychologic and other reasons it is wise not to perform it immediately before operation.

8. The decision to perform lobectomy for bronchiectasis demands an exact knowledge of the location and extent of the lesion. This cannot be acquired by ordinary roentgenograms, nor is it a matter which can be determined at operation.

The simplest and most accurate bronchographic method is the injection of the contrast material under fluoroscopic control, with careful positioning to make sure that all areas of the tracheo-

bronchial tree are filled and that the exposures are made at the optimum time. If oblique projections are made, the bronchi on both sides can be mapped out at the same time. Bronchography is best performed considerably in advance of operation, so that the lungs are free of the opaque material when surgery is undertaken.

9. Determination of the vital capacity is not necessary routinely but should be carried out in any case in which it seems indicated. Operation cannot be safely undertaken when it is less than 1,200 cc.

10. Nasal and sinus infections as well as oral infections should be cleared up before operation. Sinus infections are frequently a causative factor in bronchiectasis, and all of these conditions are invitations to postoperative difficulties. A final search for acid-fast bacilli in the sputum is also a wise preoperative precaution.

11. The best method of preparing the patient for lobectomy is to keep him in bed, with limited periods of ambulation daily, until a day or two before operation and then to permit him to be fully ambulatory. The patient who is treated in this manner comes to operation in far better physical and mental condition than the patient who is confined steadily to bed.

Technical Considerations

The tourniquet method of lobectomy has for the most part outlived its usefulness. With modern methods of preparation and anesthesia it is usually not indicated even in poor risk patients, in whom speed of operation was formerly essential. It is occasionally useful in cases in which there is considerable induration of the tissues, but it has all the disadvantages which go with crude mass ligation and all the consequences of the presence of devitalized tissue. If it is used, drainage must be continued for a long period of time, usually at least two weeks.

Intrahilar lobectomy and segmental excision[18] have practically displaced tourniquet resection in bronchiectasis. Both are based on sounder surgical principles. Segmental excision is particularly applicable to cases in which the disease is diffusely present in several lobes and in which it is necessary to preserve as much uninvolved lung tissue as possible.

The preferred approach for lower lobectomy is posterolateral, with the incision beginning paravertebrally and extending forward along the seventh interspace to the anterior axillary line. The pleural cavity is entered through the seventh interspace and the paravertebral portions of the seventh and eighth ribs are divided, which permits wider separation. In individual cases a higher or lower interspace may be used for the incision. If pneumonectomy

is to be done, the incision is placed anteriorly through the third interspace or posterolaterally through the fifth.

Opinions differ as to the position of the patient on the table. Overholt and his group[15] favor the face-down position, with the pelvis, shoulders and head suspended. Most surgeons prefer the lateral position and do not share their opinion that it is conducive to contamination of the contralateral lung. The Trendelenburg position is always contraindicated because of the risk of filling the upper lobe bronchus of the dependent lung with foreign material.

The bronchopulmonary segment is excellently adapted for surgical removal because each such segment has its own set of structures. The bronchus and the artery penetrate through the hilum into the substance and the venous channels return along the surface. The principles of individual ligation are therefore thoroughly applicable, and segmental resection is usually possible without undue difficulty. After the artery, bronchus and vein have been ligated and divided, the diseased segments are separated from the normal segments, which have previously been inflated by raising the intrabronchial pressure. The line of demarcation is thus clear, the plane of separation is comparatively avascular, and gentle, blunt dissection with the finger and thumb is usually all that is required. The visceral pleura is separated by sharp dissection, preferably after the segments are separated, when the intersegmental pleural line is clearer. Clamps are not used. The edges of the segments remaining in situ may be left unsutured or may be repaired with fine sutures, as the surgeon prefers.

In patients who have a good deal of secretion it is best to close the bronchus as promptly as possible, though seldom before the arteries are ligated. It is carefully isolated and the whole area is walled off with gauze to avoid contamination. While there are various methods of handling it, my own preference is for the following technique: A row of interrupted sutures of fine black silk is placed one interspace proximal to the proposed site of division. The ends are left long, to serve as traction sutures. A clamp is placed on the bronchus, two or three interspaces distal to the point selected for division. The bronchus is then divided obliquely, so that the posterior muscular portion is longer than the anterior cartilaginous part. The stump is treated with alcohol and then is closed with interrupted mattress sutures of fine black silk. Finally, a flap of pleura is used to cover the suture line. Penicillin is applied directly to the stump and the whole pleural cavity is flooded with a solution of penicillin and streptomycin. Two catheters are left in the pleural cavity, one anteriorly and one posteriorly, and are brought out through the ninth or tenth interspace.

This method in effect provides a double line of sutures, and thus a double protection against possible leakage. I have found that postoperative infection does not usually occur when it is employed and when it is supplemented by the direct use of penicillin and streptomycin in the cavity.

Before the operation is begun, a cannula is placed in the ankle and whole blood is run into the vein throughout the operation. The usual blood loss is from 1,000 to 1,500 cc. Even if bleeding is minimal at least 1,500 cc. of blood is given while the patient is on the table and more if hemorrhage is excessive. An 18-gauge needle is placed in the antecubital vein, for use in emergencies, and in an occasional poor risk patient a cannula in the other ankle. I can recall at least one patient in whom the administration of blood and glucose solution through all three of these channels was lifesaving.

Overholt et al.[15] prefer to operate under procaine anesthesia, with endotracheal intubation under topical cocaine anesthesia, on the ground that this method preserves the cough reflex and is associated with fewer postoperative pulmonary complications. My own preference, like that of most other surgeons, is for cyclopropane-ether anesthesia. Mousel's[17] excellent article on the role of the anesthetist should be read by all who are interested in this phase of thoracic surgery. His role is vital. The surgeon who undertakes lobectomy or pneumonectomy has all that he can do to operate. The anesthetist must be responsible for the rest. As Mousel points out, he belongs on his feet during the procedure. The incision is so draped that he can watch the steps of the operation, and can evaluate by direct observation the character of the respirations and the movements of the diaphragm, so that he can tell, before there is clinical evidence in the color, pulse and blood pressure, that tracheobronchial obstruction or mediastinal flutter has occurred or is about to occur, and can take the necessary steps to correct the situation. He must be prepared to pass the bronchoscope if catheter suction is not adequate. He is responsible for the slow decompression of the lung before the pleura is opened, for re-expansion of the uninvolved portions of the lung at intervals throughout the operation, to make certain that uninvolved bronchi have not inadvertently been ligated, and for routine bronchoscopy at the close of the procedure. A surgeon has no right to undertake thoracic surgery unless he has the services of an anesthetist who is capable of doing these things, and an anesthetist has no right to give an anesthetic for such an operation unless he can carry them out. It is not too much to say that whether a patient shall live or die on the operating

table, and afterward, depends almost as much on the anesthetist as on the surgeon.

Postoperative Care

Routine bronchoscopy, as already mentioned, is carried out on the operating table, at the conclusion of the procedure, and it is a sound additional precaution to make roentgenograms at once, to be certain that the lung has expanded properly. As Mousel[17] points out, this is an unnecessary precaution in most cases, but there is no way of determining in which cases it is essential.

Following operation, the patient is placed in the head-down position, to facilitate the ready evacuation of secretions and to favor their gravitation into the upper respiratory tract. If shock should be present, which is seldom the case today, this position is useful in combating it.

As a rule, the patient is placed on the operated side, to immobilize it and to permit free aeration of the contralateral side. Under no circumstances does he lie on the unoperated side, because of the danger of compression of the uninvolved lung and of interference with respiration. If he wishes, he may lie on his back, and he may sit up as soon as he desires. He is encouraged to change his position for himself. If he does not, it is changed for him. He is also encouraged to cough frequently and to raise secretions voluntarily. Good nursing care is invaluable immediately after operation. The nurse who sees to it that the patient changes his position, breathes deeply and coughs regularly, and who watches his color, pulse and respiration, to detect immediately any respiratory difficulties, has much to do with his prompt and smooth recovery.

Some authorities use oxygen routinely after an operation for bronchiectasis. Others believe that the best results are accomplished if the patient does his own breathing, in a normal atmosphere, on the ground that the effort encourages rapid re-expansion of the lung. Transnasal tracheobronchial aspiration is carried out at regular intervals, and bronchoscopy is resorted to without delay if there is any evidence of retention of secretions or of atelectasis which is not relieved by this simpler measure.

Transfusion is seldom necessary immediately after operation, or, if the convalescence is smooth, at any time thereafter. Infusions of glucose are given in amounts sufficient to maintain an adequate fluid balance. Physiologic salt solution is used sparingly, with due regard to the latest warnings of Coller and his group[19] concerning its dangers. If nausea is not present, as it usually is not, intravenous fluids are necessary only for a brief time after

operation, since fluids can be given by mouth without delay. Soft diet and then full diet are given as promptly as they are tolerated. As a rule, the patient is on full diet within 24 hours after operation. Like the preoperative diet, it should be high in protein and carbohydrate constituents.

Sedation is employed only on strict indications. It inhibits the cough reflex and favors the retention of secretions, particularly in older persons. Demerol is better than morphine, but all drugs of this kind are stopped as soon as possible.

Penicillin by intramuscular injection, in amounts of at least 25,000 to 50,000 units every three hours, and streptomycin, ¼ gm., also every three hours, are continued for five to seven days after operation. They are not discontinued until the temperature is normal and the pulse and respiration are within normal range.

Measures to prevent thrombosis and embolism are particularly indicated in older persons. Free movement and deep breathing are effective preventive measures, and the use of compression bandages, from the toes to the groin, is a wise precaution in older patients and in all patients with varicose veins.

The sooner the patient is out of bed, the better. He may stand, with support, the night of operation if he cannot void otherwise, and he may be out of bed, for increasing lengths of time, after the second or third postoperative day. By the end of the first week, and usually before, the patient subjected to lobectomy or pneumonectomy is fully ambulatory.

Drainage is seldom necessary for more than two or three days after operation. The catheters are not removed, however, until 12 to 24 hours after there has been a cessation of respiratory movements in the water trap, until clear breath sounds have been heard over the operated side of the chest, and until there has been x-ray evidence of the re-expansion of the lung.

While complications, particularly empyema, still occur occasionally after operations for bronchiectasis, they are no longer the rule, and the patient who is prepared for operation by the methods just described, who is subjected to an atraumatic procedure under competent anesthesia, and who is treated after operation by the regimen just outlined usually has a smooth, uncomplicated recovery and is out of the hospital within 10 to 14 days.

Lung Abscess

In chronic lung abscess the preoperative preparation, postoperative care, the anesthetic phase, and most of the technical details are much the same as for bronchiectasis. Chronic lung abscess is a condition which should not exist. If acute lung abscess is properly managed, it will not become chronic.

Classification

In the past it was customary to refer to lung abscesses as acute, subacute and chronic. Now the tendency is to classify them as simple and complicated, as suggested by Overholt and Rumel,[20] or as uncomplicated and complicated, as suggested by Moore.[20] This classification is, of course, related to duration. An abscess which has lasted less than six weeks is usually unilocular and uncomplicated. One of seven to 12 weeks' duration is likely to present such complications as multiloculation, multiple abscess-formation, spillover bronchopneumonia, gangrenous extension, and early fibrosis. An abscess of more than 12 weeks' standing is likely to present all the characteristics just listed, together with well-established pulmonary fibrosis and bronchiectasis. The abscess of brief duration is not always uncomplicated, but the abscess of long duration is practically always complicated.

The Prevention of Chronic Lung Abscess

Neuhoff and his associates[21] were the first to take the position that the majority of lung abscesses become chronic, that is, complicated, because they are not interrupted in the acute stage and the first to insist, contrary to the general opinion, that an acute lung abscess is a surgical disease. That their position was sound was apparent in the results which they reported: operation in 104 acute abscesses, with four deaths, and their later report,[22] in which they brought the number of operations to 162, with no additional deaths. When one considers that the estimated mortality of lung abscess was up to that time between 25 and 40 per cent, it is possible to appreciate the excellence of a mortality of 2.47 per cent.

Neuhoff and his group were, of course, fortunate, in that they saw patients early. The usual experience is more like Sweet's,[23] who in 125 cases saw only eight early enough for the abscess to be drained by the end of the sixth week.

Drainage of the acute abscess is not always necessary. In a quarter to a third of all cases medical measures are effective, including rest, fresh air, postural drainage, bronchoscopy and antibiotic therapy. Since these measures are not, in themselves, attended with any risk, it is fair enough to test them, perhaps for six to eight weeks, though the decision as to their effectiveness must rest on the basis of what they are achieving and not on how long a period they have been tried.

Progress must be followed from week to week, or oftener, by roentgenologic examination. The presence of a fluid level, or its persistence at the same site, is certain evidence that conservative

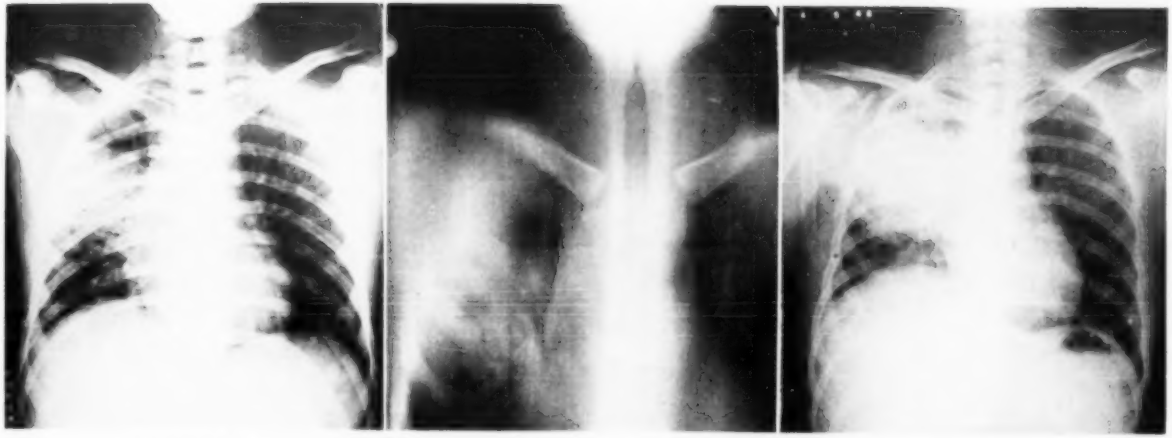

FIGURE 3 FIGURE 4 FIGURE 5

Figs. 3, 4, 5: Serial roentgenograms showing progression of lung abscess over a five month period during which the patient, a 20-year-old man, refused operation. Interruption of the phrenic nerve and various medical measures were completely ineffective. Eventually massive hemoptysis forced him to accept surgery, which, because of the spread of the disease, had to take the form of pneumonectomy instead of the lobectomy originally advised. Recovery was uncomplicated and relief of symptoms complete.

treatment has failed, even if it includes intrabronchial drainage. Any increase in the size of the cavity and persistence of, or increase in, pneumonitis, is similarly to be interpreted. An improvement in the patient's general condition, associated with radiologic improvement, is the only indication that spontaneous resolution is occurring. Deterioration in his condition, associated with radiologic evidence of progression, or with stationary radiologic findings, is an indication of the failure of conservative therapy and of the necessity for urgent surgery in the form of drainage.

The Management of Chronic Lung Abscess

However useful medical measures may be in acute lung abscess, they offer nothing but palliation, if that, in the chronic stage. Frequently progression of the disease occurs while they are being undertaken (Figs. 3, 4 and 5). Most other measures which have been proposed are no more useful. Pneumothorax is dangerous as well as ineffective. If the lung is collapsed and pulled away from the thoracic wall, the abscess may rupture into the pleural cavity and a putrid empyema may follow. Moreover, when the lung is allowed to re-expand, the chances are that the abscess will still be present. Interruption of the phrenic nerve is both illogical and dangerous; by interrupting the cough mechanism, it prevents such evacuation of the abscess as would be possible spontaneously. Thoracoplasty is a mutilating operation which contributes nothing at all to drainage of the abscess and unnecessarily collapses a large portion of healthy lung.

Surgical drainage is no solution of the problem of chronic lung abscess. Even evacuation of the purulent contents does not take care of multiple abscess-formation, chronic organizing pneumonitis, fibrosis, atelectasis, bronchiectasis, and perforation with encapsulated empyema. It does not offer any relief to the patient with persistent cough, purulent sputum, recurrent febrile episodes, secondary anemia, and loss of weight and strength. Drainage, moreover, is likely to be followed by fistula-formation which obstinately refuses to heal.

Successful drainage does not necessarily end the patient's difficulties. A persistent epithelized cavity and secondary bronchiectasis are possible, and undesirable, end-results. Another manifestation of unsatisfactory healing is the so-called lattice lung (Gitterlunge) of Sauerbruch, which can be described as the pocketing by strands of tissue of a partially healed and epithelized abscess cavity.

This is, unfortunately, the condition of many patients with chronic lung abscess when they are first seen by the surgeon. My lot has apparently been happier than that of Sellors,[24] who re-

marks, with a good deal of bitterness, that in lung abscess "the period of pulmonary destruction seems to be a jealously guarded medical prerogative." However, in my experience, too many patients with lung abscess are seen later then is desirable. The ideal plan would be to call the surgeon as soon as the internist has established the diagnosis, not necessarily to operate, but to determine, after joint appraisal, whether surgical treatment is likely to be necessary.

For a patient in the state above described only lobectomy offers a prospect of cure (Figs. 6 and 7) and pneumonectomy (Fig. 8) may be necessary because the disease has advanced beyond a single lobe. I am disposed to agree with Lindskog[25] that primary resection, without preliminary drainage, is the procedure of choice in lung abscess with multiple abscess-formation, extensive destruction of one or more lobes, secondary bronchiectasis, atelectasis and pneumonitis, repeated hemoptysis, and localized empyema. From the technical standpoint it is quite possible to evacuate the empyema and resect the involved lobe at a single stage.

When Lindskog's series of cases is divided into separate categories, the numbers are too small to be statistically significant. It is nonetheless striking that the mortality was lower, and the percentage of complete cures higher, with primary lobectomy than with other procedures. Secondary resection after previous drainage

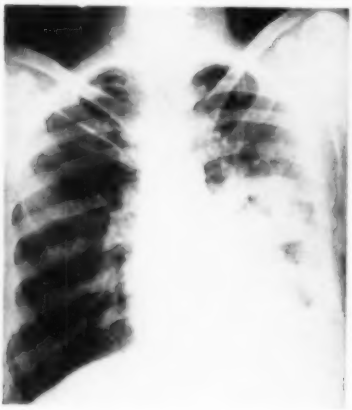

FIGURE 6

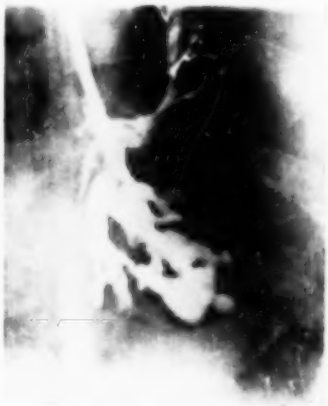

FIGURE 7

Fig. 6, 7. Serial roentgenograms showing progression of lung abscess of left lower lobe in a 44-year-old man in whom left lower lobectomy was eventually performed. Note well-established bronchiectasis in figure 7. Roentgenogram four months after operation showed that the remaining lung tissue on the left side had expanded to fill the hemithorax.

was attended with a higher incidence of late complications and delayed deaths than was the primary procedure. This might be expected. Prolonged suppuration makes secondary surgery difficult, the lung is usually densely adherent to the thoracic wall, the hilar tissue is indurated, and inflamed lymph nodes are numerous. The risk is frequently worth taking, however, for, as in bronchiectasis, the pathologic process in chronic lung abscess is irreversible. When once secondary bronchiectasis and pulmonary fibrosis have occurred, the lung is permanently diseased and only eradication of the diseased tissue offers a chance of cure.

CONCLUSIONS

In two important chronic lung conditions the rationale of surgery has been presented, certain important technical considerations have been described, and the preoperative and postoperative management and the anesthetic routine have been outlined. Adams[14] is correct when he says there is no more grateful group of patients than those who have been cured of chronic pulmonary disease by the surgical measures that are entirely practical today. Both bronchiectasis and lung abscess, however, are diseases in

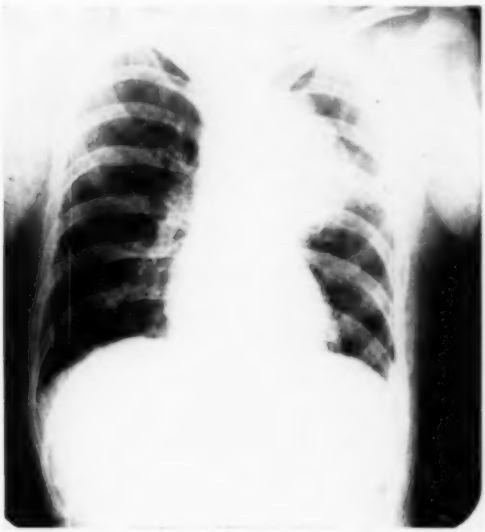

FIGURE 8: Roentgenogram of chest in 32-year-old man showing lung abscess which had become so advanced when patient was first seen that pneumonectomy was necessary. Hemoptyses had been so exsanguinating in this case that 4,000 cc. of blood had to be administered before the patient was considered even as a fair risk for surgery. Recovery was smooth and relief of symptoms complete.

which the surgeon is not self-sufficient. He cannot expect success without the close and intimate cooperation of the internist, the roentgenologist and the anesthetist.

CONCLUSIONES

Se han presentado las razones que explican el empleo de la cirugía en dos estados pulmonares crónicos importantes, se han descrito ciertas consideraciones técnicas importantes y se han bosquejado el tratamiento preoperatorio y postoperatorio y la rutina en la administración del anestésico. Adams[14] tiene razón cuando dice que no hay ningún grupo de pacientes más agradecidos que los que han sido curados de una enfermedad pulmonar crónica mediante las medidas quirúrgicas que son completamente prácticas en la actualidad. Tanto la bronquiectasia como el absceso pulmonar, sin embargo, son enfermedades en las que no basta el cirujano solo. Sin la íntima cooperación del internista, el roentgenólogo y el anestetista, el cirujano no puede esperar obtener buenos resultados.

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The Place of Social Work in a Tuberculosis Hospital*

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In a previous publication we have defined "comprehensive medicine" and made special reference to psychosocial factors in illness and treatment. The patient's problems around his illness, his recovery and his readjustment to the community present a challenge to the physician far and above his specific medical and surgical skills. In the past few decades, farsighted medical investigators have come to recognize the importance of planning for the socio-economic and emotional well-being of the patient. To do this professional groups with special disciplines have gradually been enlisted in the total treatment program. One important group is that of social work which is the subject of this presentation.

There is perhaps no professional field which has been less understood or appreciated by the medical profession generally than that of social work. In large measure this may be charged to faulty medical education, a situation which will only be changed as we approach the goal of comprehensive medical care. It is also true that the field of social work, because of its uneven development in the last 30 years, has not always been articulate enough in its interpretation to the physicians. There is, however, a growing awareness in medical education of the importance of social casework with emphasis being laid as well in premedical education on the disciplines of the social sciences.

Such acceptance as was available in the past was purely in the manipulative sense. It was felt that social workers pried into people's affairs, investigated financial status, arranged for welfare aid, wrote innumerable letters, and secured for their clients the common everyday necessities of life. The social worker combined all the features of an errand boy and just avoided the onus of a whipping boy as well.

Doctors have not been aware of the growth of social work with the need for specialization in fields of family case work, medical social work, and psychiatric social work. Such multiplicity of terms and fields has further confused physicians and has widened the gap of understanding. The psychiatric orientation which is given

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in all levels of training of social workers of whatever type has made them further suspect. This is so because most physicians are still unable to accept psychiatry, scoff at it openly, pay lip service to the field if spoken of at all, and will resent even more a non medical group which undergoes and utilizes its disciplines.

Social work from a disorganized and ill developed group has evolved into a well integrated profession with high standards, and with disciplines on a par with other professional workers. From untrained welfare workers, the level has risen to a skilled corps which can and does have an important place in social medicine.

Both in training and in practice, the medical profession has always had a highly individual approach to each patient with the doctor as the sole authority. The nurse has been accepted as a useful handmaiden at the beck and call of the physician, but the social worker has been only thought of in terms of real, practical patient's needs which play no part in the physical status. The inability of the physician to accept the ancillary services of the social worker has held back the psycho-social approach to illness and progress in comprehensive medicine. Especially in chronic medicine is there a need for the social case work approach. In a hospital setting where the patient is removed from the security of his family and enters a new type of community living with a new set of rules by which he must abide, there must be a tempering of the authoritative medical approach by the warm, accepting attitude of the case worker.

It is always interesting in a self-critical fashion, to review our own administrative attitudes regarding the patients under our care. We like best those patients that are submissive and compliant, that ask for little, are grateful for small attentions, and in short, are "good guys." We become extremely angry with the hostile and demanding personality for whom the attentions given them never seem to be enough. The food is bad, the floor is dirty, the nurse does not rub his back properly, the floor physician does not see him sufficiently, and so on 'ad nauseam.' Instinctively the administrative approach is one of counter hostility particularly when the patient continues to raise difficulties long after the incident has passed. The smiling patient is liked, the surly quickly passed by.

Disciplinary discharges are numerous while patients signing out against advice have provoked tuberculosis groups to numerous special studies. A recent study on A.M.A. or irregular discharges in the Veteran's Administration revealed that in most hospitals there were as many as 50 per cent with one hospital ranging in the neighborhood of 75 per cent. In the average hospital the discharges against advice range from 20 to 35 per cent. Studies done

to unravel the problem reveal a multiplicity of causes, all pointing to one main basis. The major attention has been paid to the physical and too little to the emotional needs of the patient. In large measure this may be due to lack of understanding, training and staff facilities.

In addition then to the staff training of physicians, there is need for a social work staff which is psychiatrically oriented. What is then the function of social work within the confines of the hospital?

Social casework is concerned with the many social and emotional problems arising out of tuberculosis and the patient's reactions to them. Its major objective and usefulness parallels that of medical care as it proceeds from the point of diagnosis through the period of hospitalization and after-care. The basic tool of this process is the relationship established with the patient through the technique of interviewing. This involves the sensitive listening and participation by the caseworker in a professional, disciplined manner. The techniques of casework have evolved from extensive professional training which draws upon the fields of sociology, economics and psychiatry. The worker brings to this relationship the kind of personality and talent that enables him to sense the patient's anxiety and feelings in general. The range of casework service is broad as it varies from helping with environmental problems to that of intensive efforts in the areas of personal maladjustment and emotional difficulties.

It would indeed be difficult to describe the many aspects of social casework in a tuberculosis sanatorium. At best we may be able to give some indication of the broad areas covered by social casework with a closer examination of some of its practices. Generally speaking, it can be said that social casework functions in relation to the client or patient, and to the community around him. There are many problems that can be worked through with the patient in an interview situation. On the other hand, the patient has many difficulties in his immediate and more remote surroundings. These can be dealt with by the social worker either by interviewing or corresponding with patient's family, community agencies and the staff that is servicing the patient in the hospital. We would like to point out that the major emphasis in social casework is the relationship between the patient and the case worker. Any productive activity must be patient centered. It means that the patient must be involved to a great extent in the thinking and planning for his welfare.

Social casework with the tuberculous patient and his family deals with the problems that he has to face in the progressive stages of his life as a patient. Thus we might say that casework should be available at the point of diagnosis, at the beginning

period of hospitalization when there are problems around adjustment, at crucial points in treatment such as surgery, at the point where the patient should be thinking of discharge and related plans for rehabilitation and also for some time after the patient has been discharged.

Let us take the first problem; that is, the patient's reaction to the diagnosis. It is rare that the individual who has just learned of his tuberculosis has no deep emotional reactions to it. Many people have considerable anxiety and, therefore, are blocked from considering next steps. Some may become extremely depressed and may, therefore, behave in a rather irrational manner. A few somehow go on as though they never heard their diagnosis; such individuals unconsciously block out this knowledge because of their inability to tolerate it. The case worker attempts to work through with the patient his feelings about tuberculosis. Such a process enables the patient to overcome enough anxiety in order to plan for treatment and for whatever assistance the family may need. In some cases all a patient will need is the reassurance that the economic problems resulting from his disability will be met by some social agency. In other cases the social work problem may be more complex because of the meaning of the disease to such patients. Thus the insecure person who has strong feelings of inadequacy will be given a chance to talk about his sense of defeat and failure brought out by his disease. The acceptance of these feelings by the social worker will mean an acceptance of the patient as an individual. There are too many variations on this complicated problem to be taken up in our limited space. It is important to point out that casework affords the kind of interview that permits the patient to bring out his many feelings and doubts into the open. The social worker does not barrage the patient with too much information, nor does he reassure falsely with cheery platitudes. Professional experience has shown that premature fact giving or pollyannish "you'll be all right" statements often create an emotional block which dams up the patient's feelings and obscures the problems of the disease. When the social worker has enabled the newly diagnosed patient to talk through his feelings about the disease, the latter can be helped with the problem of securing hospitalization.

Once the patient comes to the hospital he has many feelings about being there and about having to undergo strict routine. The social worker sees the patient immediately after he arrives at the hospital. The series of interviews during the first few weeks of the patient's stay are so carried on as to give the patient the feeling that he can talk freely about his new situation. Some patients will express a great deal of lonesomeness for their families, especially

if they are far away from home. The warmth and understanding shown by the social worker bring about some transfer of dependency from the patient's family to the worker. Thus the newly created gap between the patient and his family is to some extent filled in by the relationship with the case worker. This does not mean the beaming of sweetness and light upon the patient, nor does it mean persuading the patient to cheer up and be good. Rather, the process is one of bringing out in the open, feelings of anxiety and even anger without making the patient feel ashamed.

It is important to note in this connection that many patients have conflicts about accepting dependency upon the staff. Some patients have had a need to be successful in their social and economic activities and their illness and hospitalization have prevented them from attaining such goals. The doctors and other members of the hospital staff often notice that such patients are extremely resentful of having to lie down and to accept orders. The interviews with the social worker can bring out into the open their reasons for such resistance to the hospital regimen. The patient is, therefore, better able to examine the basis for his feelings, and also finds it easier to accept dependency because of the tolerant attitude of the social worker.

There are many other reasons why a patient may find it hard to adjust to the hospital in the beginning or at any other time during his stay. There are, for instance, economic and environmental problems in the patient's family. The social worker is unique in the field of human relationships insofar as he works with the family and community. It often happens that the worker has to discuss at some length with a patient's wife, husband, parent or any other meaningful relative the problem of the disease as it has affected them. The wife and mother may need some specific help in securing relief for the family, or medical care for one of the children. Perhaps she may find the new burden of being both father and mother too difficult. Her relationship with the social worker becomes a source of support and security to her and at the same time allays the anxiety of the patient. Here again it may be said that the example given was a relatively simple one, and that often the problems in the family become much more complex and, therefore, much more difficult to handle. Often a mate or even a relative may become rejecting because of the patient's disease. If such a person is willing to talk it through with the social case worker, he or she may be helped to develop less threatening attitudes toward the patient. In some instances the patient has been a client of some welfare agency that has taken responsibility for him. As he develops economic needs or shows other problems that are in the province of that agency,

the hospital social worker interprets such problems to that organization. The problems requiring contact with agencies are too numerous to mention. To emphasize then, the social worker serves as a liaison between the patient in the hospital and the community, which has the effect of reducing the anxiety of the patient and preserving to a great extent his sense of still belonging in the world around him.

A great deal has been said about the many problems that a patient has in the hospital. Experience has shown that many patients have deeply rooted anxieties which need alleviation. The social worker is part of a team working in conjunction with the doctor, the psychiatrist, the vocational counsellor, the occupational therapist and other auxiliary workers. The responsibility for helping patients with emotional difficulties is that of the total hospital. Therefore, the attending physician as the authoritative figure in the hospital setting, has the total responsibility for the patient. If this physician has sufficient time to spend with the patients, and if he has received some psychiatric training and had done work in social science, then theoretically, he would be able to work with the patients along the lines needed. With this background he should then be able to refer patients to social agencies. The reality, however, is that a physician is not equipped to do social work because of his lack of training. While the doctor has the responsibility of developing relationships with the patients which will help them in their anxiety about their illness, he has to accept the specialized services of social work.

On the other hand, the social worker has rather a key position insofar as he does not symbolize the authority or power of the physician. This relative absence of administrative power gives many patients more freedom to discuss openly their complaints, anxieties and hostile feelings about the hospital, its treatment and staff. It is well known that a patient must be able to bring out into the open some of his simmering hostilities if he is to get help with his difficulties. In some cases a psychiatrist will do the treatment as the problems have to be dealt with in terms of their unconscious derivation. In the majority of cases, however, the social caseworker is equipped by technique, training and ability to conduct the kind of interview that will be helpful to the patient. It must be borne in mind that the social worker has definite and concrete goals when he works with a patient's feelings. The case presented later in the paper illustrates this form of activity.

There are some critical situations in a patient's life in the hospital. The whole problem of surgery is a complex one fraught with considerable anxiety for the average patient. Collapse therapy often represents mutilation, disfigurement and the removing of

essential parts of the body. Patients react to the prospect of this surgery with varying degrees of anxiety depending on their personal makeup. Some patients are so distraught that they are ready to leave the hospital rather than undergo such a procedure. Verbal reassurances such as "Don't worry. You'll be all right," and many other statements aimed at alleviating the patient's feeling usually fail. The patient must be given a chance to talk about his anxieties concerning treatment. The psychiatric literature is replete with well documented cases of unconscious death wishes. It can readily be seen that such individuals may unconsciously look forward to surgical procedures as the attainment of such desires. The social worker can give the necessary time in one or a number of interviews to explore with the patient his feelings about this treatment. Some patients will be better able to understand their resistance as part of their vanity about their bodily appearance. Others will obtain relief from just ventilating their anxiety about pain, mutilatin and death. It has been noted that repression of fear often brings about unfortunate results after surgery.

The social worker has a stake in the rehabilitation of the patient. Since rehabilitation is not only concerned with vocational training but rather with the social, economic and emotional adjustment to the post-sanatorium life it is important to understand the patient's personality, his fears and his dependency needs. The social caseworker has had training in the field of mental hygiene as well as practical experience in interviewing patients so that he is able to arrive at an understanding of the individual. In our experience we have found that a discussion of vocational training often leads to a definition of personality problems. Thus some of the patients who ask for re-training are helped to see that their basic difficulty was not the kind of work that they did, but rather their inability to cope with their employers or colleagues because of certain emotional difficulties. In other instances it was noted that the patients expressed need for a new vocation arose out of his anxiety about leaving the protective atmosphere of the hospital. The social worker can make a real contribution in the planning for vocational re-training by working closely with the vocational counsellor so that there is a true understanding of the patient's total needs.

It was stated earlier that the social worker is concerned with the post-sanatorium existence of the patient. Where the hospital social worker is unable to see ex-patients because of the time and geographical factors, some interpretation should be made to an agency and community to which the patient could turn for help. The National Jewish Hospital has recognized the need for continuing care and therefore, has set up social work departments in two major cities. The social workers within the hospital maintain

close contact with the social agencies throughout the country. Before a patient is ready to leave the social worker interprets to the agency in his community the rehabilitation and other needs that he presents, and discusses with the patient what is available to him in his community.

The social worker plays an important role in helping the patient plan for discharge. It is erroneous to think that all patients will eagerly accept the prospect of leaving the sanatorium. Many patients, after a long period of hospitalization, have learned to accept their dependency and concurrently have developed many fears about resuming normal living. Some patients even give up former relationships such as husband and wife because they have substituted for this relationship the strong parental figure of the hospital. These people and many others will balk at plans for physical and other kinds of rehabilitation. The trained social worker can help the patient to bring out into the open what his real anxieties are and in some cases the patient develops enough insight to go on with a plan for rehabilitation. Thus we find the patient who has considerable difficulty about making up his mind about what kind of training to take finally admit that he is blocked by his unwillingness to sally forth from the hospital into a competitive world.

The following is a presentation of a case which illustrates the role of the case worker in the total treatment of a rather difficult patient: Lucille had been curing tuberculosis for four years and would be ready to leave the hospital in a few months if all went well. Everybody in the hospital knew Lucille. Most of the medical staff groaned with a fair amount of good nature when her name was mentioned. Patients shook their heads over her behavior but on the whole they were sympathetic and friendly toward her. There had been ups and downs throughout her hospitalization, however, as the time of her discharge approached, she seemed to have more severe upsets and to be under tension most of the time. She became outspoken, abusive and demanding toward those in authority when the tension spilled over. She complained continuously and as soon as one demand was met she countered with another. She would test the patience and tolerance of doctors and nurses over and over again. Although she was able to reason with astute accuracy about the causes of her actions and to recognize that her behavior was defeating the goal for which she was fighting by provoking an immediate discharge, she did not seem to be able to mobilize enough strength to do anything about it. Her appetite became ravenous and she violated her diet in spite of her recognition of the dire consequences to herself. Her attitude was, "I am as I am because of the things that have been

done to me. I am not responsible for my behavior and therefore you must not blame or punish me."

Lucille was a young woman 26 years old, dark complexioned with deep set blue eyes, of medium stature and inclined to be plump. In periods of upset she gave the impression of being a dumpy disheveled little girl. She expressed herself unusually well and had a nice sense of humor. The wall of sophistication, however, was brittle and broke with the slightest pressure. She was in constant search of praise and had need of immediate tangible rewards.

Lucille was born and reared in Texas. Her mother died of tuberculosis when she was four. She had been placed with relatives even before her mother's death because her mother had been too ill to care for her. She was the youngest, by seven years, of seven children. Her mother was in her forties and her father was in his late fifties when she was born. There would seem to be serious question that she was a wanted child. Although her father did not die until years later, he paid little attention to her after her mother's death and she spoke of his having deserted her. At first, after her mother's death, Lucille lived with various relatives. They felt that she was a difficult child and by the time she was six, she was placed in an orphanage. From there she was placed in a foster home. She remained in this home for eight years and then had to leave because the home was broken following the foster father's death. She was placed in a second foster home and lived there until she became ill with tuberculosis. When her foster parents learned that she had tuberculosis they were unwilling for her to return to their home. This repetition of family rejection and feeling of being unwanted was a devastating experience, reactivating all the unhappiness and loneliness of her childhood. For some time after her hospitalization she was depressed, she felt deserted and that she had little reason to get well. In addition to the economic and emotional deprivations, Lucille has had to cope with serious health problems all of her life. She had surgery at six months for obstruction in the esophagus and feeding was difficult from the beginning in terms of this condition. It was learned that she had diabetes when she was six.

Intensive casework service was begun at the time the patient's conflicts with administrative and medical authority became acute and immediate discharge was being considered. Regular weekly hour interviews were scheduled with the social worker and in addition, Lucille was invited to consult with her social worker whenever she felt the need to do so between these interviews. A positive relationship between Lucille and her social worker was established rather quickly in terms of her extreme need for sup-

port. She was at the same time suspicious of her social worker and tested the worker's acceptance of her repeatedly.

There was a consultation between the psychiatrist and the social worker soon after intensive casework service was begun for the purpose of (1) securing a diagnostic personality evaluation on the basis of the information available and the patient's presenting behavior; (2) establishing the goal of treatment; and, (3) deciding the type of casework service the patient could use effectively. It was recommended by the psychiatrist that the worker should assume the role of a mother figure, i.e., a warm, giving person. Patient's behavior was an acting out of her wish to be treated as a child, to be loved and cared for. Such a relationship is necessary to such a dependent person in order to reenforce her in difficult spots. It was felt that the goal of casework service could not go much beyond this in terms of the retardation of emotional development because of earlier deprivations. It was felt at this time that continuation of some supporting figure would be necessary after her return to the community.

In addition to the psychiatric consultation between the social worker and the psychiatrist, this case was discussed in the regular psychosomatic conferences with the medical and other interested administrative staff. The discussion was lead by the psychiatrist. Members of the staff having direct contact and responsibility for patient's care presented material for the discussion. The social worker presented a psychiatrically oriented social history material and a review of the problems. The doctor having direct responsibility for her care then presented the medical history and administrative problems arising in terms of behavior in the hospital. He raised questions concerning management. Other doctors volunteered information on the basis of their contacts and observations. The dietitian gave a review of her difficulties with the patient in relation to diet violations and special demands. The purpose of the conference was to further the understanding of the underlying dynamics responsible for the presenting disturbing behavior in the patient which might result in more understanding management. This was accomplished up to a point, however, it would not be logical to assume that there would be sufficient carry over from a single conference of this kind to create a sustaining attitude which could offset the irritations created in the day to day stress of handling her behavior. Individual conferences between the social worker and the physician followed to discuss further the psychological implications of her behavior and an attempt to enlist a more positive identification with her. There were conferences also from time to time between the social worker and dietician who decided that dietary problems might be resolved

more easily if there were fewer restrictions imposed and more responsibility was given to Lucille in handling the situation. She responded positively to the change.

Lucille did lean heavily on the social worker during this period. She used the interviews to discuss her plans for work following her discharge from the hospital and to express her anxiety around having to leave. She gradually recognized that she could express her feelings of resentment against the hospital, her doctors, and the world in general to the social worker without fear of retaliation or criticism. Thus some of the tension and anxiety was reduced. This support, reenforced by a modified handling by her physician, rather quickly brought about a noticeable change in her behavior.

The last major upset occurred about two months after intensive casework service started. Lucille became very anxious when she was moved to the ward from which patients are discharged. She voiced many complaints, stating that she had been moved ahead of other patients that should have gone before her, that there were mice in her room, that the room was so damp she was having arthritis. She finally became ill and took to her bed, she complained of severe pain in her shoulder and lower back. When the social worker saw her she insisted that nothing was being done for her pain, that she was being treated as if she were a mental case. She was refusing to go to meals saying that she was too ill. Later the social worker conferred with her physician who expressed considerable irritation over her demanding attitude, "she feels she is the only patient I have. When I ask her in fairness to consider the welfare of other patients, she becomes abusive." Her background was reviewed with him, her present needs and attitudes were discussed particularly as they related to authority and authoritative father persons. When he saw her the next time he spent some time discussing her work plans following discharge, commenting on the excellent report he had heard about the results of her vocational test and so on. The effects of this interest were spectacular. There were continued upsets but they were of shorter duration and less intense.

In working through plans for work following discharge there was a real opportunity for strengthening Lucille's confidence and building morale inasmuch as she had demonstrated ability to do competent work and had shown responsibility on the job prior to her illness. She had worked steadily after completing business training. The vocational aptitude tests given in the hospital vocational rehabilitation process indicated that her skill was above average in the clerical field. This strength was utilized in her general rehabilitation in the hospital by providing opportunity, through the hospital bulletin, for recognition of her ability. And

later, as her work tolerance advanced, she was given work in the hospital medical office. About this she commented, "I feel as if I am among the living again and I can hold my head high."

As time of her discharge approached she struggled against leaving by making a strong plea to be retained as an employee. She hoped to have her living quarters there also. Giving recognition to her continued dependency need in considering plans for rehabilitation, arrangements were made for her to live in a local convalescent home with continued supervision from the hospital medical and social service. When the transfer was first made she offered many complaints against the home but there were no serious upsets. This transfer accomplished a physical separation but she held tenaciously to the hope of being able to continue her relationship with the hospital by a work placement there. When the time arrived that she was ready for full time employment, there were no suitable openings for work in the hospital. The social worker informed her of the situation with a great deal of apprehension since her ability to accept this final and complete separation would be the acid test of her readiness to function independently. She did not receive this disappointment without considerable feeling but she did face it without any serious upheaval. Two weeks later she was able to tell her social worker that it had been hard to take and that she had been depressed, "it seems like an awful kick in the pants now," she added, "but I guess it is about time I stop being a parasite and grow up a little."

This activity of the social worker in conjunction with the staff demonstrates the various areas in which the case worker functions, and also the sensitive handling in the interview situation. This patient was an extremely disturbed individual whose background of deprivation helps us to understand the dynamics of her personality. Her inability to make relationships other than those of a hostile and dependent nature presented a challenge to the administration. It was difficult for the staff to cope with the bitter effrontery of this woman. Knowing that the patient's personality was determined by a number of factors in her life situation the total staff accepted the need for her treatment by the doctors and by the social worker. The latter by her warmth and sensitivity to the patient's feeling was able to establish the kind of warm, amiable relationship that gradually overcame her bitter suspiciousness. We could see progress in her attitudes towards the staff as the casework unfolded. For one thing, her hostility was greatly lessened and therefore she had less need to violate hospital regulations and in the process destroy herself. Secondly—she was helped to accept some plan of rehabilitation. The final outcome was some modification of her dependency so that she could transfer to the

Ex-Patient's Tuberculosis Home and to continue planning for economic self-sufficiency.

SUMMARY

1) The medical profession has never been able to completely accept social work. It has been able to see social case work only in the manipulative sense but never in the sphere of therapy.

2) The place of social work in a tuberculosis hospital is carefully detailed beginning with its place in the acceptance of the diagnosis, proceeding to hospital admission, and continuing throughout the hospital stay and through vocational rehabilitation and discharge.

3) The patient presents problems at each stage of his hospital stay, and with each must have the help of an understanding hospital staff and especially the social service department.

4) A case is presented to demonstrate the many problems involved and the functioning of the social case worker in the situations presented.

SUMARIO

1) La profesión médica, no ha aceptado completamente todavia, el trabajo social. Se ha observado el trabajo social solamente en el manejo de los casos, pero no como tratamiento.

2) La importancia del trabajo social en un hospital de tuberculosis, está demonstrada con la aceptación del diagnostico, al ingreso al hospital, durante la estadia en el mismo, durante los cursos de vocación y rehabilitación y cuando es dado de alta.

3) Los enfermos presentan problemas en cada etapa de la estadia en el hospital, y en cada uno de ellos es necesario la ayuda inteligente del personal del hospital y especialmente del servicio social.

4) Para demonstrar los múltiples problemas involucrados y el trabajo del auxiliador social, se ha presentado un caso como ejemplo.

CORRECTION

In the article entitled "Factors Influencing the Outcome of Streptomycin Therapy of Pulmonary Tuberculosis" by Dr. William B. Tucker, Minneapolis, Minnesota, published in the December issue of "Diseases of the Chest," the legend given for Figure 1 (page 722) was incorrect. The following is the correct legend:

"*Fig. 1:* (J.V.) Young World War II Filipino veteran, pulmonary tuberculosis and tuberculosis of right anterior 4th rib, healed following excision. Density over right second anterior interspace is of iodized oil injected into sinus tract.—*Fig. 1a:* January 16, 1947: progressive pulmonary disease just prior to start of streptomycin therapy, classed as "unmixed" acute (see text).—*Fig. 1b:* June 10, 1947: marked x-ray improvement at end of SM therapy (2 gms. day for 120 days).—*Fig. 1c:* August 3, 1948: very marked x-ray improvement, 18 months after start of therapy. Sputum negative by culture for over a year. No relapse since."

College Chapter News

THIRD ANNUAL POSTGRADUATE COURSE TO BE PRESENTED IN PHILADELPHIA

The Third Annual Postgraduate Course in Diseases of the Chest sponsored by the Pennsylvania Chapter of the American College of Chest Physicians and the Laennec Society of Philadelphia will be held at the Warwick Hotel, Philadelphia, Pennsylvania, April 10-14, 1950. Registration for the course is now open and applications will be accepted in the order in which they are received. A coupon for enrollment in the course will be found in front advertising section, page xii.

SAN FRANCISCO POSTGRADUATE COURSE

The California Chapter of the College, in cooperation with the University of California Medical School and Stanford University School of Medicine, presented a postgraduate course in recent advances in diseases of the chest in San Francisco, December 5 through 9, 1949. The course was presented in the University Extension Building of the University of California. Eighty physicians were registered for the course. Out of state lecturers in the course were Dr. Andrew L. Banyai, Associate Clinical Professor of Medicine, Marquette University Medical School, Milwaukee, Wisconsin, and Dr. Herman J. Moersch, Associate Professor of Medicine, Mayo Clinic, Rochester, Minnesota. Drs. Seymour M. Farber and William L. Rogers, San Francisco, served as chairmen of the course.

NORTHEAST BRAZILIAN CHAPTER

The Northeast Brazilian Chapter held its annual meeting in Recife, Pernambuco on the 19th, 20th and 21st of September. The following program was presented:

"Cytological Diagnosis of the Carcinoma Bronchio Genico,"
Humberto Menezes, M.D.

"The Cooperation of Patients in a Campaign Against Tuberculosis,"
Aldo Vilas Boas, M.D. and Laurenio Lima, M.D.

"Differential Diagnosis in Mass Chest X-ray Surveys,"
Jose Rollemberg, M.D.

"Extrapleural Pneumothorax,"
Jose Rollemberg, M.D.

"Streptomycin in the Treatment of Tuberculosis,"
Herodoto Pinheiro Ramos, M.D., Alcides Ferreira Lima, M.D.
and Jose Brasiliense de Holanda Cavalcanti, M.D.

"The Place of the Dispensary in the Campaign Against Tuberculosis,"
Jose Rollemberg, M.D.

ROCKY MOUNTAIN CHAPTER

The Rocky Mountain Chapter is planning a meeting to be held in Denver, Colorado, on Tuesday, February 21. The program will consist primarily of an x-ray conference. The chapter also plans to hold a rehabilitation conference in the spring.

ILLINOIS CHAPTER

The Illinois Chapter held a meeting at the Congress Hotel on January 13 at which time the following program was presented:

"Experiences in the Treatment of Pulmonary Tuberculosis with Lucite Plombage,"
Charles K. Petter, M.D., F.C.C.P., Waukegan.

"Pneumoperitoneum Combined with Streptomycin in the Treatment of Pulmonary Tuberculosis,"
Earl Gilbert, M.D. and George C. Turner, M.D., F.C.C.P., Chicago.
Discussant: William J. Bryan, M.D., F.C.C.P., Rockford.

"The Use of PAS in the Treatment of Tuberculous Empyemata,"
M. R. Lichtenstein, M.D., Paul Alfano, M.D., Chicago
and H. Vernon Madsen, M.D., F.C.C.P., Ottawa.

College News Notes

Dr. J. Edmond Bryant, Evanston, Illinois, Chief, Division of Chest Diseases, Provident Hospital, visited Haiti, British West Indies, and Colombia, South America. In Port-Au-Prince, Haiti, Dr. Bryant visited Dr. Auguste Denize, a Fellow of the College.

In Bogota, Colombia, Dr. Bryant visited the recently built San Carlos Hospital for the treatment of tuberculosis. Dr. Pedro Jose Almanzar is the Director of the hospital and Dr. Pablo Leiva is in charge of the Department of Surgery. While in Bogota, Dr. Bryant visited with Dr. Carlos Arboleda Diaz, Governor of the American College of Chest Physicians for Colombia and Director of the Hospital Santa Clara. He also visited with Dr. Rafael Jose Mejia, a Fellow of the College and Director of the Tuberculosis Hospital LaMaria.

Dr. Bryant reports that considerable interest has developed among the members of the College in Colombia for the First International Congress on Diseases of the Chest to be held at the Carlo Forlanini Institute, Rome, Italy, in September 1950.

Dr. Bryant was honored by election as a corresponding member to the Sociedad Colombiana de Tisiologia.

University Hospital of the New York University-Bellevue Medical Center has named Dr. Frank R. Ferlaino, New York City, assistant clinical professor in the department of industrial medicine and assistant attending physician.

Dr. Frank S. Dolley, Los Angeles, California, lectured at the University of Montevideo, Uruguay, in December. On December 9, Dr. Dolley lectured on the "Diagnosis of Tumors of the Mediastinum" at the Hospital Fermin Ferreira, and on December 10 his lecture was on the subject of "Extrapleural Pneumothorax: Indications, Technic and Results" presented at the Hospital Maciel. Dr. Dolley also participated in the Second Argentine Congress on Tuberculosis held in Cordoba, Argentina, November 28-30, 1949.

Dr. Benjamin Gasul, Chicago, Illinois, addressed the meeting of the Maricopa (Arizona) County Medical Society, held in November, on "Congenital Heart Lesions."

At the sixth session of a medical postgraduate seminar in Bemidji on November 1, Dr. John F. Briggs, Minneapolis, discussed the treatment of heart disease. The session was part of a seminar sponsored by the University of Minnesota Medical School, the Minnesota State Medical Association and the Minnesota Department of Health.

Dr. Edward J. Nagoda, Tucson, Arizona, addressed the Pima County Tuberculosis and Health Association on "The Modern Drugs for Tuberculosis."

Dr. Chevalier Jackson, honorary professor of bronchoesophagology at Temple University School of Medicine, Philadelphia, has received an honorary fellowship from the Royal College of Surgeons in Edinburgh, Scotland.

The Japanese Bronchoesophagological Society was formally inaugurated in Tokyo on November 20, 1949. Dr. Jo Uno, a Fellow of the College, was elected as the Society's first President.

THE JAMES ALEXANDER MILLER FELLOWSHIP FOR RESEARCH IN TUBERCULOSIS

The New York Tuberculosis and Health Association announces that a fellowship will be available from July 1, 1950 to June 30, 1951. The fellowship is designed to support a qualified medical investigator who will devote full time to a research project with a definite bearing on tuberculosis. Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the pursuit of the work. It is preferred that such laboratory or clinic be located in New York City.

The stipend will be $5,000.

Application forms will be supplied on request to the New York Tuberculosis and Health Association, 386 Fourth Avenue, New York 16, New York, and must be submitted in duplicate not later than March 1, 1950. Announcement of the award will be made in April 1950.

BINDING FOR COMPLETED VOLUMES

We are pleased to announce that The Book Shop Bindery, 308 West Randolph Street, Chicago, Illinois, will produce a well-bound volume at as low a price as possible for those members and subscribers who wish to preserve their issues of DISEASES OF THE CHEST. They will bind the six issues of Volume XVI in the best grade of washable buckram with gold stamping on the spine and the member's or subscriber's name in gold on the front cover. Please send the six issues to Chicago by express or parcel post prepaid with check or money order for $2.75 made payable to The Book Shop Bindery. The bound volumes will be returned with transportation prepaid by the bindery.

Obituaries

Herbert Arthur Burns

1883-1949

For more than 40 years, Dr. H. A. Burns so successfully combatted contagious diseases of humans and animals that he became one of America's outstanding benefactors of humanity. Following graduation from the University of Minnesota School of Medicine and completion of an internship in 1909, he engaged in private practice until 1912 when he was appointed epidemiologist for the Minnesota State Board of Health. There he played an important role in controlling epidemics of diphtheria, typhoid fever and the like, and participated effectively in the control of tuberculosis. Soon after discharge from military service with the rank of captain in April 1919, he was so interested in tuberculosis that he resigned from the State Department of Health and became supervisor of tuberculosis trainees among veterans. From 1920-28, in addition to this work he devoted extra time to the Lymanhurst School, the Parkview Sanatorium, and the Medical School of the University of Minnesota. In 1928, he was appointed assistant superintendent of the Minnesota State Sanatorium, and one year later became superintendent and medical director. He promptly converted the sanatorium from a domiciliary institution to a hospital. Consultants were appointed in all phases of medicine and surgery, and a fine chest surgery service was established. Between 1935 and 1939, the sanatorium buildings were remodeled and the institution was converted into one of the finest physical plants in the state.

Dr. Burns' thorough knowledge of epidemiology stood him in good stead. In 1931 he appointed a full-time epidemiologist to the sanatorium staff. This was probably the first time in the nation that such importance was given to the epidemiology of tuberculosis by a sanatorium. Arrangements were made for the distribution of free tuberculin to the physicians of the 44 counties his sanatorium served. He also provided an x-ray interpretation service for these physicians as well as free consultations with the members of his staff. The preventorium of the sanatorium was converted into a hospital for contagious cases. A special contagious disease technic was put into practice throughout the institution to protect personnel from tuberculosis. Surveys were conducted among American Indians and Dr. Burns was influential in having the federal government erect a 117 bed building for tuberculous Indians on the sanatorium grounds in 1934. The same year he published the results of a tuberculosis survey which he had personally conducted in the nine state institutions for the mentally ill and defective. This, which was the first survey of its kind on a statewide basis in this country, revealed the most serious tuberculosis situation in the state's population.

Dr. Burns' ability to solve the most difficult problems had long been

recognized. Therefore, in 1942 he was relieved of duties at the State Sanatorium and appointed chief of a newly created tuberculosis control unit in the Division of State Institutions. Immediately he sought out all the cases of contagious tuberculosis among patients and personnel alike and arranged for isolation centers.

His loyalty to the nation was so great that he volunteered for military service and was accepted with the rank of major in 1944. On separation in 1946, he returned to the direction of the tuberculosis control unit of the State Division of Institutions. The crowning event came in 1949 when the legislature appropriated funds for a building where practically all mentally ill patients in the state could be hospitalized under Dr. Burns' direction.

He had always been in excellent health. However, in January, 1948, a mass which was recently palpated in the region of the left kidney, proved to be a hypernephroma. Before metastasis attained clinical proportions, he died from a massive brain hemorrhage on July 8, 1949.

Throughout his life, Dr. Burns exhibited energy and endurance which far surpassed that of most men. His mental capacities seemed unlimited. He was so enthralled by professional activities that work was recreation. He lived far in advance of his time and thus frequently outlined procedures and discussed plans which could not materialize until many years later. He had a large store of knowledge on a wide variety of subjects, both in and out of the field of medicine.

For many years he held membership in the National Tuberculosis Association and the American Sanatorium Association (later Trudeau Society). He was a staunch member of the American College of Chest Physicians and served on the Committee on Chest Disease in Penal and Mental Institutions. He was an exceedingly valuable member of the Committee on Tuberculosis of the Minnesota State Medical Association and a similar committee of the American School Health Association. He published more than 20 papers.

Dr. Burns pioneered several projects which have been adopted as standard procedures in tuberculosis control. He always struck squarely and forcefully at the tubercle bacillus and never tolerated slipshod or short-cut methods, or those based on theory for which there is no satisfactory premise. He never went on tangential excursions, but hewed straight to the line. His entire professional life was in a direct course toward the goal of eradication of tuberculosis. This enabled him to make so many outstanding accomplishments of worldwide value in a lifetime of 66 years.

Abstracted from Journal-Lancet, April 1949.

Bruce Hutchinson Douglas

1892-1949

The passing of Bruce H. Douglas, Health Commissioner of Detroit, Michigan, left a void in public health and tuberculosis control activities in this country which will long remain. He had prepared himself so well for his chosen field of action, his value was so enhanced by so many years of experience, and his sphere of influence so large, that it will be long before this nation shall look upon his like again.

Born in DesMoines, Iowa, on August 26, 1892, he received the degree of Doctor of Medicine from Rush Medical College, University of Chicago, in 1920, after which he went to Detroit as an intern at Harper, Children's and Herman Kiefer Hospitals. There his work was so outstanding that at the end of the year he was appointed Assistant Chief Resident Physician at Herman Kiefer Hospital for the year 1921-22. His unusual ability, trustworthiness, sincerity and large store of information were recognized by the officials of the William H. Maybury Sanatorium, who selected him as Assistant Medical Director in 1922 and promoted him to the medical directorship in 1923 and to the superintendency in 1924. Thus, within four years from graduation in medicine, he was superintendent of one of America's best tuberculosis sanatoriums.

In this capacity he became an expert physician and promoted every aspect of tuberculosis work from case finding, accurate diagnosis and every worthwhile method of treatment and prevention of the disease. In this position he taught numerous physicians, nurses, social workers and others the fundamental methods of tuberculosis control, not only in the individual patient, but also in the community and the nation at large. After serving the Maybury Sanatorium for 11 years, nine of which he was Superintendent, he was chosen as Comptroller of Tuberculosis by the Detroit Health Department. From 1933 to 1941 he made one outstanding contribution after another and developed original techniques in tuberculosis control. His addresses were heard by large numbers of persons. His published articles were read everywhere and his good advice was followed. Throughout the years, Dr. Douglas became highly qualified for the health commissionership of Detroit and was awarded this position in 1941. At that time, his fine qualifications as a teacher caused him to be appointed Professor of Public Health and Preventive Medicine in the School of Medicine, Wayne University, and Lecturer on Public Health in the School of Public Health, University of Michigan. Thus, during the last eight years of his life, he held the highest positions in public health and education afforded by his city.

In tuberculosis, Dr. Douglas' work and influence were superb. Upon entering this field in Detroit more than a quarter century ago, he immediately became active in local and state organizations. From 1933 to 1938 he was President of the Michigan Tuberculosis Association. Through-

out the years he was an influential member of the National Tuberculosis Association which later became the American Trudeau Society, and was elected to the presidency of the National Association in 1941. In these two organizations he was a valuable member of numerous committees and for several years was on the editorial board of *American Review of Tuberculosis*. He held fellowship in the American Medical Association, American Public Health Association, American Association for the Advancement of Science, American College of Physicians and was certified by the American Board of Internal Medicine.

He was on the governing board of many organizations such as the American Cancer Society, American Red Cross, National Foundation for Infantile Paralysis, Visiting Nurses' Association, Detroit Cancer Research Institute, Detroit Council of Social Agencies, Boys' Club of Detroit and the Economic Club of Detroit. His membership included numerous community organizations, such as the Girl Scouts of metropolitan Detroit, Detroit Interracial Committee, Detroit Youth Committee, Detroit Civil Defense and Disaster Committee. Thus, his sphere of influence extended far beyond routine tuberculosis and public health activities.

In times of war, Dr. Douglas also played his role. He was a member of the Students Army Training Corps in 1918 and served as a private in the medical enlisted reserve in 1917-18. In 1942 he was appointed Surgeon, United States Public Health Service Reserve Corps.

Dr. Douglas was tranquil, composed and kind. At the same time he was firm and always manifested the courage of his convictions. These qualities, together with his vast store of information, accomplishments, high regard for opinions of others, truthfulness, honesty, sincerity and unquestioned forthrightness won the confidence of all who knew him well. At the moment of his accidental death, August 11, 1949 the health of the citizenry of Detroit was dependent upon him and his co-workers. Numerous organizations were counting on his support; the public health world was looking forward to more of his advice and contributions to knowledge. Of Dr. Douglas it can be truly said, that he is not dead who continues to live in the hearts and activities of men.

Norman William Heysett

1904-1949

Dr. Norman W. Heysett, staff member at the Irene Byron Sanatorium for the past four years, Fort Wayne, Indiana, died on September 6, 1949. He was born in Baldwin, Michigan and attended high school in Ludington. Dr. Heysett received his degree of Doctor of Medicine from the Indiana University School of Medicine.

After leaving college, Dr. Heysett practiced general medicine in Hart, Michigan for eight years. Dr. Heysett had held membership in the American Medical Association, the Indiana Medical Society and the Allen County Medical Society. He was a Fellow of the American College of Chest Physicians.

Dr. Heysett is survived by his wife, Alice Seybolt Heysett, and a sister, Mrs. Frank B. Ritchey, Fort Wayne.

Jerome V. Pace, M.D., Governor for Indiana.

Book Reviews

INVITED AND CONQUERED, by J. Arthur Myers, Ph.D., M.D., Minn. Pub. Health Assn., St. Paul, Minn., 737 pages, 356 illustrations, 1949, $6.50.

This engrossing book, written in the author's customarily pleasing style, and dedicated to one of the most persistent yet unsung foes of the disease, is particularly timely this centennial year in documenting all the significant contributions and developments in tuberculosis in Minnesota during the past 100 years. Every incident and detail of anti-tuberculosis work, from the earliest pioneer days in 1659 through the most recent diagnostic technics of photofluorography and therapeutic procedures such as pneumonectomy are presented in chronological sequence. More than 1,900 persons who have played some role in the conquest are accorded their proper place. Pictorially also, in addition to the photographs of 245 anti-tuberculosis workers, 120 illustrations and 14 graphs enhance the value of the book. It is a monumental and much needed documentation of medical history, correlating activities within this state with the advances in tuberculosis throughout the world.

First is traced the initial infection of the Indians from the time of Radisson's first landing, in 1659, through the final stages of the New Ulm massacre in 1862. Then the story is told of immigration to Minnesota of people from all over the country and world, all of whom migrated here for the climatic cure of their tuberculosis. Koch's discovery of the tubercle bacillus only slowly was accepted as proof of contagiousness of the disease. It influenced greatly the diagnosis of tuberculosis as did also the introduction of the clinical thermometer in 1867, discovery of tuberculin in 1890, use of the x-ray after 1895 and the bronchoscope in 1898. Another chapter captivatingly tells of the early influx of doctors and the organization in 1853 of what is now the Minnesota State Medical Association, the creation and early history of the Minnesota Department of Health, the birth and foundling years of medical education culminating in the establishment of the University Medical School. Rounding out the first part are two chapters concerned with early mortality of human tuberculosis, and such early efforts to control the disease as reporting cases, quarantine and sanatoriums.

In part two are reviewed both the lesser and epochal advances in tuberculosis especially as related to Minnesota persons, organizations and institutions. Of consuming interest are three chapters discussing pioneers, former Minnesotans and the state's debt to other people. No detail is too small, no incident too trivial, no worker too obscure to be unmentioned in the historical progress against tuberculosis.

Among the institutions and organizations whose development have conditioned and paralleled advances in tuberculosis have been the University Medical School, the Minnesota Board of Health, sanatoriums and others. Here is the full story of the growth of the University Medical School from one small structure and five non-teaching faculty members in 1893 to its present size and 541 teaching faculty members this year. No less intriguing and important, so far as tuberculosis is concerned, is the story of veterinary medicine and the eradication of tuberculosis in cattle in Minnesota. Even more intimately identified with eradication of human tuberculosis is the State Board of Health. This too, is a fascinating epic of progress from conflict with other agencies to develop-

ment of a special Tuberculosis Control Division and eight branch offices throughout the state. How Minnesota's sanatorium situation developed from not a single bed for tuberculous patients in 1900 to 2,200 beds for tuberculous patients in the State Sanatorium and 14 county institutions provides here engrossing reading for anyone.

Even with discord between official and lay anti-tuberculosis organizations rife early in this century, there ultimately emerged an effective Minnesota Public Health Association. Since 1924, when the present personnel took the reins, this association has energetically and unremittingly employed every method and device to control the disease. Most effective and helpful were the tuberculin testing, tuberculin distributing and finally the mass mobile x-ray unit and tuberculin testing campaigns. Unique in Minnesota's contributions have been accreditation of counties and schools in reducing the incidence of tuberculin reactors.

No such volume is complete without an evaluation of accomplishments to date, and an entire chapter is devoted to this topic. Comparisons of the mortality of tuberculosis and the numbers of earlier and present tuberculin reactors in various groups, states and the United States are discussed and graphically presented. In fact, this whole chapter is a remarkable testimonial to the effectiveness of the work of the innumerable persons, groups, agencies, organizations and institutions mentioned in preceding pages. Also of indispensable value is the final chapter of mileposts in tuberculosis eradication. Here are listed in almost outline form all the major events from 1659 to date in the history of Minnesota's fight against tuberculosis.

This book is truly a masterpiece of research, and a basic contribution to the historical and tuberculosis literature of the state, if not the nation. Certainly, no one interested in the history of either Minnesota or tuberculosis could afford to neglect this book.

RADIOLOGIC EXPLORATION OF THE BRONCHUS, by S. Di Rienzo, Chief of the Radiology Department of the Institute of Cancer, The University of Cordoba, Argentina. Published by Charles C. Thomas, Springfield, Illinois, 1949.

Occasionally one is privileged to review a medical volume which is a work of art in addition to a book of factual medical material. The author has succeeded admirably in his purpose of demonstrating abnormalities in bronchial anatomy and physiology by radiographic means. There are 466 figures in 319 pages and the pictorial method of presentation makes for interesting reading.

The bronchographic image of the normal bronchus is clearly explained. The anatomic characteristics are indicated by the calibre, outline and branching of the bronchus. The broncho-physiologic details are demonstrated by the filling rhythm, the bronchial tone and the mechanism of the muscular sphincter as well as the respiratory variation in calibre, the peristaltic waves and the effects of cough.

The technique of injection of contrast media is outlined in detail beginning with the preparation of the patient and ending with the positions best suited to fill various lobes and segments. The type and technique of anesthesia is described meticulously. The author recognizes that there are occasional hazards including anesthetic reactions, hemorrhage and toxic reactions to the contrast media. The measures necessary to reduce these hazards to a minimum are discussed.

Tomography of the bronchus is considered a valuable procedure not only for the knowledge gained but because there is no contraindication and no instrument or chemical substance is introduced. The author believes and rightly so that the relative merits of tomography, bronchography and bronchoscopy can never be determined since these methods complement each other and are not interchangeable. Tomography is especially indicated in those cases where the disease is in the trachea, primary bronchi or main lobar bronchi.

Bronchopulmonary malformations are considered important because of their unsuspected frequency and varied forms. The malformations that make extrauterine life impossible are unimportant and are primarily of interest to the pathologist. The remainder frequently are not recognized unless superimposed infections supervene. These include lung agenesia, air cyst, alveolar agenesia, cystic bronchiectasis and bronchial diverticulum.

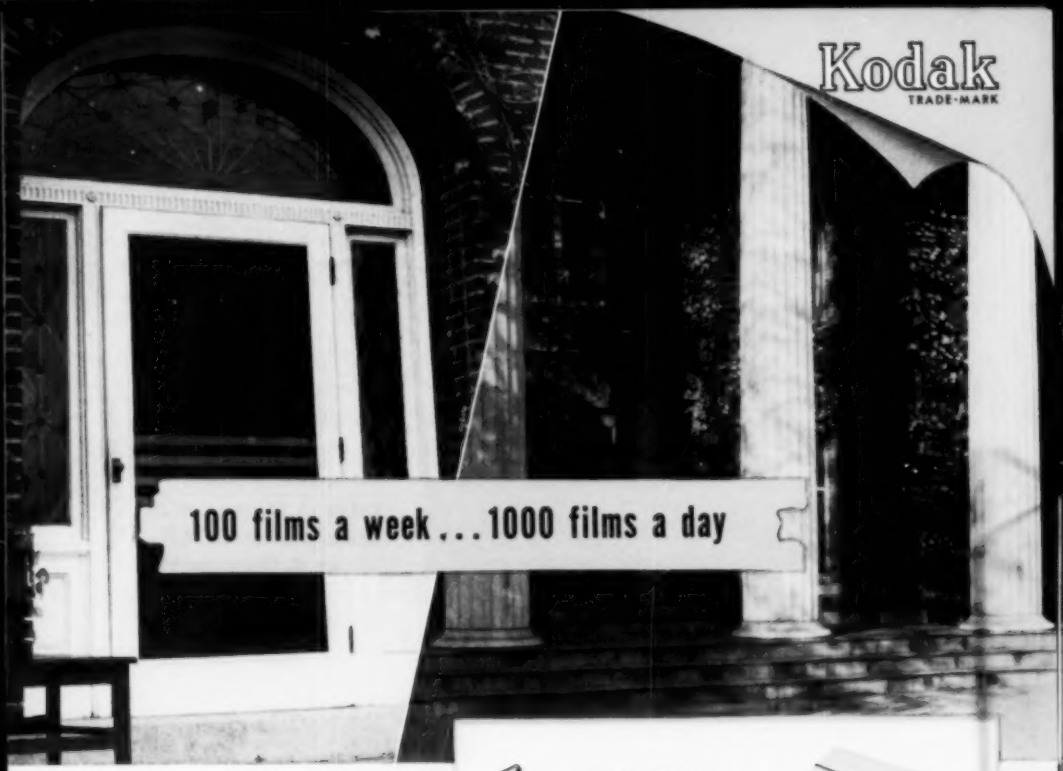
The relative importance of infection, bronchial obstruction, atelectasis and pulmonary fibrosis in the etiology of bronchiectasis has been widely discussed. The author believes the basis of acquired bronchiectasis is the development of obstruction in the terminal bronchi with the development of micro-atelectasis. The obstruction in the terminal bronchi is caused by infection, edema and secretions. The obstruction causes difficulty in expiration of air which is sharply intensified on coughing and bronchiectasis results. The author's explanation is rational but still does not eliminate infection and weakening of the bronchial wall as the initiating factor in certain cases. At times, bronchial obstruction may precede infection but in many cases infection antedates the obstruction.

In emphysema and asthma the principal change is in bronchial dynamism. The opaque substance is not "sucked in" by the fine branches during respiration but remains mostly in the medium and large bronchi. The author further states that the opaque material has a tendency to form constant images instead of transient and changeable images, which is normal. The calibre of the secondary branches is smaller than normal, indicating spasm which reduces their lumen and dynamism. There is a scarcity of foliage.

Bronchography, tomography and bronchoscopy complement one another in the diagnosis of bronchial carcinoma. The author believes tomography should precede bronchography and that bronchography should precede bronchoscopy. The author does not believe bronchography need delay surgical intervention provided strict bronchographic technique is observed. Bronchography in lesions affecting only fine bronchi and small areas is probably useless. This type of examination is most beneficial where the carcinoma begins in a first or second order bronchus. The author emphasizes that radiologic exploration does not give images of absolute diagnostic value. It only gives information of the degree of canalicular patency and the characteristics of the obstruction of the bronchial branches.

The value of bronchography in hydatid cyst and pulmonary suppuration is discussed.

The student of diseases of the chest will be delighted with this book. The reproductions of roentgenograms are marvelous and the publisher deserves special praise for the excellent printing. A complete bibliography for each section is included. This volume is highly recommended to all physicians interested in diseases of the chest.



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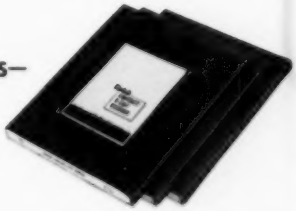

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